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Eugene O'Dunne

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We are living in an age of industrial accidents in which thousands of persons are daily killed or crippled. Workmen's compensation laws, in which Maryland was the pioneer, have swept over the country and exist in some form in perhaps every State of the Union. They are founded on the principle of making industry bear the burden of its human toll.

The demand for rapid transit is met by the railroads, electric surface cars and subways, the automobile and the aeroplane. More persons are killed by automobiles than in the world's wars. Nearly every accident resulting in injury or death is the basis for some claim for compensation, and, if not paid, it generally results in the filing of a law suit.

Every law suit for personal injury requires medical testimony as to the proximate cause of death or injury or the nature and extent of the injuries. This has vastly multiplied the work of lawyers, increased their incomes, and caused some branch of the medical profession to be engaged in almost every case.

The medical profession in all its specialized fields is thus called upon by the law to make its contribution to the cause of justice.

Courts are merely the tribunals of organized society for the dispensation of justice according to the law of the land. The medical man as well as the lawyer should always have a

clear concept of this fundamental fact, and neither should
allow his zeal to close his eyes to this basic truth. It calls
for the same degree of honesty which should obtain in the
daily transactions of commercial life—often none too rigidly
observed in either field—but it is a reproach to commerce, to
the legal and medical professions when the system in its
operation falls short of honesty and fair dealing.

THE LEGAL SUMMONS

Whether you be medical expert or mere practitioner, when
summoned to court, you must obey that summons. It is
equivalent to the command of your King, whatever the form
of government may be. It has the sanction of sovereignty
behind it. It is organized society calling upon you in the
name of the law, much the same as the present draft. Any
excuse you may have, you may present after appearing there,
but not by means of remote control from the operating room
or elsewhere. The summons left with you by the Sheriff is
not a mere invitation to have tea with lawyers and the Judge
and Jury, in case you have nothing more pleasing on hand.
No. "R.S.V.P." is attached thereto, because you have no
choice as to acceptance. You are ordered there at the time
and place specified, and you *must attend*, or be in contempt
of Court and liable to a fine or imprisonment for failure so
to do.

Lack of this knowledge has cost some doctors substantial
money, loss of reputation, loss of temper, loss of dignity, and
afforded much amusement to jurors and spectators.

THE JURY SYSTEM

The tribunal established by society in the judicial branch
of government for the redress of personal wrongs and the
protection of human rights is the Court, and an agency of
the courts is the jury system. The jury system has long
been a subject of debate. It has been condemned and
praised, perhaps beyond its deserts in either instance. It has
been wisely used and grossly abused. This accounts for much
of the rhetoric that has been expended upon it. Only lately
Judge Hanify of the Superior Court of Boston said:

"I venture to say that a few years ago none of you would have thought that a situation could arise in the world whereby men would be dragged from their homes, tried by a tribunal, and next morning brought before a firing squad.

"No jury system is in operation where there is a dictator. So long as you have the jury system, your rights will be protected—your rights of free speech, to worship as you please and to live. What good are those rights if not protected by the right of trial by jury?"

The jury has long been part of the judicial machinery, and not even advanced thought has suggested its complete abolition. There is increased demand in labor circles for its further extension and much outcry against attempts at its restriction. Without holding a brief for either side, it is well that we understand something of what it is, because it is to a jury of "his peers" that the medical man must talk in his attempt to contribute the learning of his profession to the cause of justice.

THE JURY PERSONNEL—WOMEN

A jury is composed of twelve honest men selected to apply justice according to the law of the land. They need not be wiser than the twelve Apostles, and some of them may be fishermen. Theoretically they are subject to the draft law as the triers of fact, as men of common experience, citizens, white or colored, male or female—a cross section of the community. Local laws generally regulate their ages. In Maryland, the age requirement is twenty-five to seventy, with many provisions as to exemptions from jury duty based on a theory of public convenience, such as school teachers, Federal and State employees, election officials, the deaf and the blind, but not necessarily the dumb. In practice many of the most intelligent citizens find some way to get excused.

In some enlightened States women serve on juries. At this time in Maryland we have not the benefit of their *intuition,* said to be an attribute of the angelic kingdom. We still rely on *evidence,* as established by the testimony of human witnesses. Time was, in the Proprietary period of early Maryland history, when this was different. The earliest all-woman jury in the new world sat September 26, 1656, at Charles' Gift,* the oldest building in Tidewater Maryland near Lusby, Calvert County, Maryland. During Oliver Crom-

* Charles' Gift is now owned by Hulbert Footner, the gifted author.

well's time it was for a short while the Capitol of Maryland. It was at Charles' Gift that the Great Seal of Maryland was lost, and it has not since been found. Here also eleven women sat as jurors to try Judith Catchpole for the murder of a newborn infant while crossing from England, but they found Judith not guilty. The finding may have been that she had not given birth to a child.

Time does not permit a discussion of whether Maryland's judicial branch of government in now restricting jurors to *men* lost the crown jewels with the Great Seal, or whether the chivalric spirit of Maryland's early sons wanted to shield women from contact with some of the sordid things of life. With the more complete emancipation of women, including suffrage under the Nineteenth Amendment, they stand side by side with men in commerce and industry, and will some day sit side by side with them in the jury box, as well as on the Bench.

Women soon will be flying planes, dropping bombs and shooting rifles with unerring accuracy. The task of the medical man on the witness stand will be more difficult then than now, when he is called upon to *persuade* a woman (if her intuition should lead her to think otherwise than he does), as to the nature and extent of injuries or what was their proximate cause!

Remember always that until you have women jurors, you can expect to have a jury of only average intelligence. Some jurors now may not be able to read or write, and their understanding of the English language may be extremely limited. It will be your function to so impart your scientific information as to find lodgment and understanding in the minds to which it is addressed.

PREPARING TO TESTIFY

Well, how can you best serve your country, not in the field or in the clouds, but on the witness stand? Primarily by a thorough training in your profession, which we presume you have had before reaching the witness stand. It requires more than this, at least in many instances. A careful lawyer prepares each case before trial, as an actor studies every part played by every other actor in the drama. Every trial in

court is a human drama. The medical man too often makes
a farce of it by being wholly out of harmony with the cast.
The case may require special preparation at his hands, close
scientific reading of the latest medical literature. He must
be prepared to stand cross examination by rough and ready
trial lawyers, sometimes none too delicate in the handling of
witnesses, especially if they are *ponderously pompous*.

In Hans Zinsser's "Biography of R S (As I Remember
Him)" he says:

"Doctors may be roughly divided into physicians who know a lot but
can't do anything, and surgeons who can do a great deal, but don't know
very much."

CREATE AN IMPRESSION OF HONESTY

The medical man on the witness stand must be not only
honest in fact, but must impress the jury that he is honest; that
he is desirous of being helpful as to those subject matters of
science about which he is asked; that he is impartial in the
contribution of his information, and that he is not trying at
all hazards to support the contention of the side which may
have called him. This should be done in a manner polite,
in a form as simple as the subject permits, in a tone audible,
not requiring repetition, and in language capable of being
understood by men of average intelligence and without tech-
nical training.

When Dr. Henry Rowland, then Professor of Physics at
Johns Hopkins University, was on the witness stand in the
Federal court testifying in his suit against the electric power
companies (for $10,000 as his fee for expert services as a
physicist), he was cross-examined by the celebrated Joseph
H. Choate of New York, who asked him "who is the greatest
physicist in America?" Professor Rowland, without a mo-
ment's hesitation, replied, "I am." President Gilman, of
Johns Hopkins, later asked him why he gave that answer.
Professor Rowland smilingly replied that it was a little
embarrassing, but he was *under oath* at the time and had no
alternative! Always let your answers reflect the *truth*.

To again quote from Dr. Zinsser's book:

"The average integrity of the medical profession is perhaps a little higher
than the population as a whole, but not high enough for *euthanasia*."

TRANSLATE MEDICAL TERMS FOR LAYMEN

The value of a teacher in any school depends upon his ability to impart his information to the class before him. He must get on a level with them. Here is where nearly all medical men hopelessly fail in the courts. Their minds are full of medical jargon. They know only medical terminology. They talk only the language of science. They may correctly express their thought, but it might as well not be expressed. The jury has no idea about what they are talking. The medical expert may be proud of the smoke screen in which he has enveloped the subject, but the jury is looking for the road which continues on the other side of the cloud of smoke. That is the path on which they are honestly trying to grope, to reach a terminal somewhere yonder, but they know not where. Unless you can help direct them to the right destination you are hindering justice, rather than helping it.

In Dr. Hugh Young's biography just published appears the word "Dibromoxymercuryfluorescein" (mercurochrome). There are perhaps a few of the simple men who sit on the jury who might not know exactly what this means, should you use that term in your testimony on the witness stand. It might be slightly confusing even to some judges! If that is the only word known to you which accurately describes what you have in mind, perhaps you could break it down into some of its constituent parts and feed it to the jury on the installment plan—and thereby make yourself more useful as a witness, even if you appear to be less scientific. Simplicity is an element of greatness.

If the patient has inflammation of the fluid sac and bony structure of the left collar bone and shoulder joint, why tell our simple honest jurors that he has *"traumatic arthrosteitis of the left acromioclavicular joint with inflammation of the left subacromial bursa,"* as we lately heard from the witness stand?

Vladimir Horowitz, the celebrated pianist, had to cancel his November 13, 1940 concert and postpone it until January 15, 1941, based on the testimony of a physician that he was convalescing from *traumatic tenosynovitis of the flexor digitorum sublimis and profundis muscles at the metacarpophalangeal joint.* Horowitz had a badly injured finger!

If the lawyer asks you what was the cause of death in a given case, therefore, do not ask him does he mean "the *proxima causa mortis?*" And if for the sake of peace the lawyer says, "Yes, I want to know the *cause* of death," do not tell the jury that the victim "died of *edema of the brain following cerebral thrombosis or possible embolism following arteriosclerosis combined with the effect of gangrenous cholecystitis,*" as did one of your profession who nearly got a juror in trouble with the court when he exclaimed, "Well, I'll be damned!" The Judge remarked that *ordinarily* he would not tolerate language of that kind from jurors in his Court, but the language of the doctor was equally offensive, and added that the juror had so aptly expressed the thought that was running in the mind of the Court that he believed the provocation sufficient to completely obliterate the offense.

Cryptic Explanation of Concussion.—In a case recently tried before me some of the Hopkins' experts seemed to have some difficulty as to just how to make a jury understand what constituted "concussion of the brain," and why they thought the patient had sustained this character of injury. Their explanations were by no means satisfying. Later, a little Irish doctor from South Baltimore took the stand. He was the family doctor for the plaintiff who was suing for the injuries sustained. The bland lawyer for the insurance company started to cross-examine him, and, in a patronizing way, said, "Doctor, perhaps *you* can explain what you medical men mean by 'concussion of the brain'?" The little red-haired doctor looked at the jury of farmers and said, "Did you ever take an egg and shake it violently until the yoke and the white were scrambled together? Well, that would be concussion of the brain. If the egg is fresh and healthy, the yellow might later settle down, and the white separate. If it is aged, or if the hen has sat on it too long, it might remain permanently cloudy. *That* I would liken to concussion of the brain." The jury brought in a verdict of substantial damages for the plaintiff. The jury knew something about eggs!

POLITENESS AND DIPLOMACY

Should the High Sheriff leave you a summons at your office or home some evening and you be scheduled for some im-

portant operation at the hospital in the morning, then by the use of a little politeness and diplomacy you generally can call either the lawyer, or if necessary the Judge, and make arrangements for having your testimony taken at an hour not to conflict with your operation, and still be in time for its use at the trial. This is not to be accomplished by insolence and insubordination, either in correspondence or phone messages, as I have known medical men to resort to, but by politeness and diplomacy.

Dr. William F. Bowers, in his book "Charm and Personality," illustrates what I mean in the story he tells of the plumber who was sent to a lady's apartment to make some repairs at an hour when it was thought she was downtown. On opening the door he found her submerged in the bath tub. He immediately withdrew with the exclamation, "Excuse me, Sir!" Dr. Bowers says *politeness* was illustrated in the "excuse me," and rare *diplomacy* in his emphasis on the word *"Sir."*

COMPENSATION FOR EXPERT TESTIMONY

In various States of the Union the laws differ. In some jurisdictions expert testimony can be forced from medical men the same as from ordinary witnesses and without compensation for it. In other jurisdictions the result of special study and long training is regarded much the same as private property, not to be taken, even in the public interest, without due compensation.

Where this rule prevails, a medical man on the witness stand may decline to answer a question calling for expert testimony, on the ground that no financial arrangement has been made with him for compensating him for that character of information. He may waive his right and consent to answer expert questions without compensation, if he cares to do so.

Contingent Compensation Improper.—No medical expert should have his compensation dependent upon the result of the case. This is known as a "contingent fee," dependent upon success of the litigant. Such an arrangement has a tendency to warp the judgment of the witness who has a personal and pecuniary interest in the verdict. It lays him

en to the impeachment of his integrity, oftentimes *justly*

Compensation for expert testimony is a legitimate source
revenue to the medical expert. Care must be had not to
low employment to bias your judgment. Medical men have
rought much odium upon their profession by practically
lling themselves to the side which employs them. Some-
mes their bias is conscientious, but that they *are* biased is
ll too apparent.

The "battle of the experts" is no uncommon sight in
ourts where the fight is for large stakes. The Thaw trial
as such a case. Appeal was made to Dr. Adolf Meyer,
ead of Phipps Clinic at Hopkins. He consented to examine
Thaw and to testify in New York as to his opinion on the
question of Thaw's sanity, but stipulated that he would not
accept compensation therefor from either side. Few reach
hat altitudinal record. It is not necessary to gratify legal
or medical ethics that one should do so, but such record
lights are seldom forgotten.

CONCLUSIONS

When summoned, come to Court and register your attend-
ance. If you desire to claim exemption from such draft as
an "expert," with whom no financial arrangements have been
made, make your plea to the Court when asked for *expert*
testimony. If disallowed, give your testimony. You are also
at liberty to waive your claim for exemption if you are so
disposed. No compensation to the medical witness, expert
or otherwise, should be *contingent* on the result of the case.

Avoid bias as well as the appearance of it. Make con-
scientious preparation for the efficient contribution of your
technical information to the cause of justice. Give it in such
form as to be readily grasped by lay jurymen, in language
understandable by them. Be polite and diplomatic in your
relations with court and counsel, irrespective of how they
seem to treat you.

Thus, and thus only, can you be of genuine value to the
administration of justice.
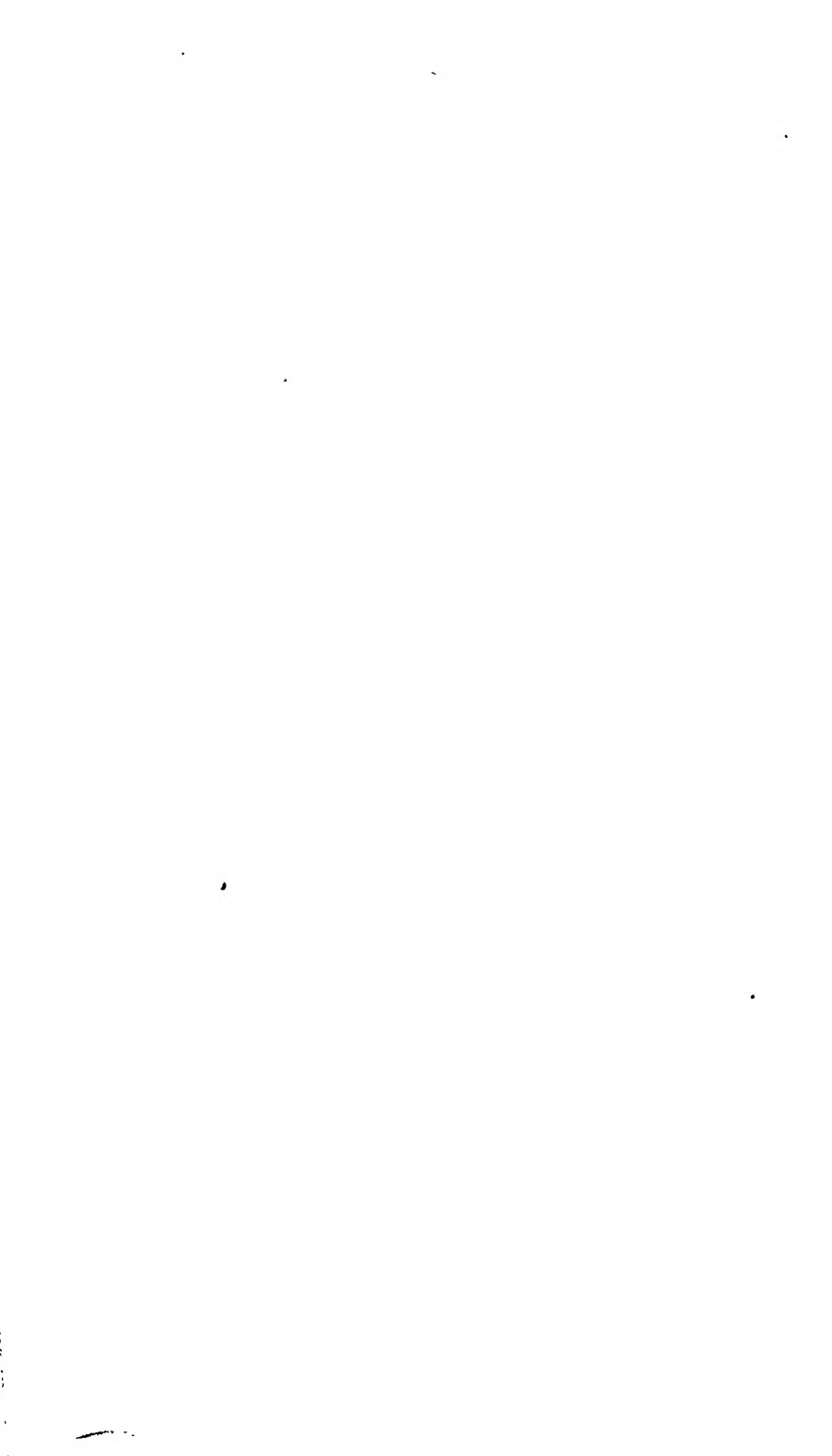
THE MEDICOLEGAL ASPECTS OF MENTAL DISEASE

Manfred S. Guttmacher, M.D.

Chief Medical Officer of the Supreme Bench of Baltimore

THE recognition by the Courts of the legal importance of mental disease can be traced back to the earliest periods in English law. The supposition that insanity has become a defense against a crime only with the birth of yellow journalism is erroneous. In the Middle Ages, madness was a complete defense* against a crime, and by the eighteenth century psychiatrists walk across the pages of the reports of famous British trials much as they do today. Moreover, the medical experts were then badgered by pettifogging legalists with many of the very same tricks and formulae that the professional descendants of these gentlemen employ today.†

Because of the overshadowing dramatic interest of psychiatric testimony in criminal trials, there is a misconception that the chief sphere of contact between psychiatry and the law is the Criminal Court. As a matter of fact, it is in the Civil Courts that most of the medicolegal psychiatry is done. These civil cases fall into three large groups: (1) problems of adjudication of the insane and their commitment to mental hospitals; (2) mental disorders following personal injuries; and (3) the mental competency of individuals to make binding contracts. In this last category occurs the specialized contractual problem of wills, and the annulment of marriages and the granting of divorces on the grounds of mental disease.

The Courts are not interested in the diagnosis and classification of mental disorders. Their focus is directed to the individual's intellectual capacity and his behavior. Their chief concern is in determining the *degree* by which these functions deviate from the legally established norms. As a

* Edward III (1326–27).
† Doctor Monroe, in Earl Ferrer case.

consequence, the writer does not plan to discuss specific psychiatric disease entities, but to deal primarily with sanity and insanity, concepts which are legal rather than medical.

MENTAL DISORDERS AND CRIMINAL RESPONSIBILITY

The Tests of Responsibility.—In criminal trials in which the issue of insanity has been raised, the Court's interest is almost exclusively confined to the psychological problem of cognition. If the traverser had at the time of his crime sufficient intellectual capacity to distinguish right from wrong and to realize that, if apprehended, he would suffer punishment for his act, he is considered in most states of the Union a responsible agent. This so-called *knowledge test* is the chief test of criminal responsibility in every jurisdiction but New Hampshire. The Courts of that state held in a remarkable decision written in 1866, that there is no adequate legal test of mental responsibility, but that if a criminal act is committed by an individual with a diseased mind, and if the act can be logically considered a symptom of his disease, the traverser is not guilty by reason of insanity.

Seventeen states have, in addition to the knowledge test, the *irresistible impulse* as a cause of irresponsibility. This concept was first enunciated in Ohio in 1834. It holds that an individual is not responsible, even though he recognizes the wrongfulness of his act, if his will be so weakened by disease that he is unable to resist the morbid impulse that motivated his misconduct. Many of the severely neurotic criminals who are driven by compulsions to steal, set fires, etc., come within the meaning of this Act. The law of the irresistible impulse is difficult to interpret accurately because the distinction between those who can and those who cannot resist their pathologic drives is extremely difficult. Nevertheless, the concept is of great value because it introduces into the law recognition of the fact that psychopathologic behavior is not produced by disordered intellect alone, but may result from diseased emotions and their control.

It is a relatively simple matter to find glaring faults in these legal definitions of mental irresponsibility. The *knowledge of right and wrong test* has its foundations in an English decision rendered more than two hundred years ago. Great

medical leaders like William White and brilliant legal minds like Benjamin Cardozo have for years pointed out the unscientific character of these tests. However, no one has come forward with a legal measure of responsibility that is any more satisfactory. Further, it is the writer's belief that there never will be developed a legal definition of mental responsibility that is adequate to cover all types of criminal behavior. Far sounder from many angles is the New Hampshire rule, which employs no formal test of responsibility but merely considers whether the defendant is mentally disordered and whether his act resulted from his disease. If insanity pleas were heard by Judges or Juries specially trained in psychiatry, as has been advocated, undoubtedly the New Hampshire rule would be excellent. But the field of psychiatry is bewildering to most laymen, and without some rules to guide them, untrained individuals are in danger of growing hopelessly confused.

There are well qualified physicians who refuse to participate in criminal trials because they believe that they cannot give sound medical opinions when the established legal tests are employed. They point out the fact that Judges do not agree on the basic meaning of the tests. There have been frequent disagreements as to whether "right and wrong" refers to *moral* or *legal* right and wrong. They fail to realize that, today, many Judges are aware of the inadequacy of these tests and are very sympathetic toward the plight of the medical witness. As a matter of fact, many of the most experienced and conscientious psychiatric experts do not attempt to view a criminal case through the narrowed focus of "the right and wrong" test. Because of their special training, they can determine whether the defendant is suffering from a mental disorder and whether his criminal act has resulted from it. After they have reached their own decisions on these issues, they translate them into the framework of the current legal formulae, and by so doing, they have carried out the spirit of the law.

Expert Testimony in Criminal Trials.—Even more unsatisfactory than the legal definition of mental irresponsibility is the method of presenting expert testimony in criminal trials. Psychiatrists are presented in criminal trials as

rank partisans rather than as men of scientific objectivity.
This same criticism can be leveled at the system of expert
testimony in Civil Courts as well. Only, in the Criminal
Courts, the violence done to justice is more impressive be-
cause there a man's liberty and even his life depends upon
the testimony of the expert.

The common practice of having the prosecution and the
defense *each employ* its own paid psychiatrist is pernicious.
The expert is generally carried away by the importunings and
the enthusiasm of his employers. It is as difficult to remain
objective and fair when surrounded by a staff of eager lawyers
as it is to remain neutral when seated in a college cheering
section. A few noble souls can withstand the pressure, but
they are likely to miss the mark by bending backward. The
writer painfully recalls how some years ago the inherent
viciousness of our present system of partisan testimony was
brought home to him. While waiting to testify in a civil
case, he was seated next to one of the country's leading
neurologists, who had been employed by the other side. The
case was discussed and we found ourselves in complete agree-
ment. My colleague then remarked, "I believe under the
present setup it is our duty to present cases in the most
favorable light possible to our respective sides, without doing
actual violence to the truth." He smiled wryly and added,
"The good Judge will never realize the unanimity of our
opinions." This was no medicolegal shyster who took cases
on a contingent fee basis. This was a man of high medical
ideals who was then the president of his local medical society.

One of the most capable and respected members of the
Maryland Bar recently told the writer in the presence of two
Judges that he would employ only medical witnesses who
testified the way he felt they should testify; that he could not
afford to employ *impartial* experts.

A growing dissatisfaction with partisan medical testimony
in criminal trials has given rise to a constantly increasing use
of psychiatrists *officially appointed* to represent the Court.
These men examine the accused and testify as neutrals, as
fair and impartial in their presentations and conclusions as
the Judge himself. Today, in many of the larger municipali-
ties there are permanently organized medical departments

serving the Courts. Their chief function is the psychiatric study of defendants, either before trial or before sentence is imposed. The pioneer work of this kind was begun by Dr. William Healy in 1908 in connection with the Juvenile Court of Cook County, Chicago. The most important development in extending the use of neutral psychiatric examiners in the Criminal Courts was the passage of the Briggs Law by the Massachusetts Legislature in 1921. This law makes the psychiatric examination of major offenders *mandatory*. The examinations are carried out on behalf of the Court by the Massachusetts State Department of Mental Diseases. Reports of the psychiatric findings are filed with the Judge, the Prosecutor and the Defense Counsel prior to trial. If either side is dissatisfied with the conclusion of the Court's expert, it may employ its own psychiatrist to testify. Dr. Briggs informed the writer that this practice has been very rare since the law went into effect, because both the jury and the Judge put much more reliance in the opinion of the neutral psychiatrist than in that of the partisan expert. Michigan has recently enacted a statute much like that of Massachusetts. In Colorado, whenever a plea of "not guilty by reason of insanity" is made, the defendant must be sent to a state hospital for observation. In six other states this practice may be followed if ordered by the Court.

Statutes providing for the appointment of neutral experts to examine a defendant whose mental condition is under question have been recently enacted in several states. In three states the appointment of neutral experts by the Judge of the Criminal Court is mandatory as soon as an insanity plea is made. In ten states the appointment of such experts is discretionary with the Court.

In the writer's opinion, statutes should be enacted making it mandatory to appoint neutral experts in all cases—civil and criminal—in which problems requiring expert medical opinion arise. The state medical societies could annually submit to the Courts a panel of qualified experts from which the trial Judges could choose. Compensation of such neutral experts might be made a part of the Court costs. By such a system, the law could be honestly enlightened and medicine would regain its reputation of trustworthiness and its self-respect.

Lawyers, as a group, are even more conservative than doctors. Many intelligent members of the legal profession to whom the writer has expressed his views on the importance of the neutral expert are horrified at the idea of having a physician assume a judicial role. Yet, it is psychologically interesting how in their minds ermine trappings can magically transform a mediocre lawyer into a Judge possessing superior wisdom. The writer recognizes and respects the intricacies of sound judicial work. He realizes that men must have specialized training and experience in order to perform it efficiently. But he does not see why it would be impossible to find physicians with the same inherent intellectual ability and judicial qualities, who could advise and even share with the Judge the responsibility of reaching just decisions in medicolegal matters.

In order to lay a foundation for such an advance, it is highly desirable that our medical schools should put the same emphasis upon medicolegal training as is done in European universities. The reforms so badly needed in the medicolegal field must, in the writer's opinion, come from an aroused and united medical profession. The lawyers are too well satisfied with the status quo; they have been playing the game by these same rules for two centuries. Precedence and inertia form for them impenetrable barricades.

Insanity Verdicts in Criminal Trials.—The issue of insanity may arise very early in criminal cases. An individual may be so mentally disordered that he is incapable of aiding in his own defense. In such cases, the defendant is sent to a mental hospital where he remains until he has recovered sufficiently to stand trial. In criminal trials, in which there is a plea of "not guilty because of insanity," two clear-cut issues arise: the mental condition of the accused at the time of the crime, and his condition at the time of the trial. If he is declared insane at the time of the crime and insane at the time of trial, he is found not guilty because of insanity and is committed to a mental hospital until he has recovered. However, the defendant may be found to have been insane at the time of his crime and to have regained his sanity during the interval before his trial. Under this anomalous verdict, the defendant—sometimes a murderer—is in most jurisdictions

automatically freed. This is the so-called *temporary insanity verdict*, and has done more than anything else to bring medicolegal psychiatry into disrepute. Scientifically, such a state as "temporary insanity" may exist; its occurrence, however, is very rare. The writer is able to recall only two such instances in the two thousand defendants he has examined for the Criminal Courts of Baltimore during the past ten years.

The general public has little interest in science. It rightfully feels that its protection is the primary purpose of the Courts, and that it is a sorry state of affairs when a murderer is permitted to go scot-free because of some medicolegal technicality. The sound scientific position in this matter is that an individual, who was so mentally disordered a short time before that he irresponsibly committed a crime, needs a period of prolonged institutional observation before it is safe for him to go about in the community. Fortunately some states have tackled this practical problem realistically.

In ten states, an individual found not guilty because of insanity goes automatically to a state hospital. The question of whether he has regained his sanity by the time of the trial is not raised. In California and Utah, he must remain in the hospital for a minimum of one year, and in Indiana, for a minimum of two years. Indubitably this type of procedure should be adopted in every jurisdiction.

Sixty years ago the Courts of England directly attacked this important question. The old verdict of "not guilty because of insanity" was discarded and the formulation "guilty but insane" was substituted for it. This form of verdict is philosophically unsound, since an insane man lacks sufficient capacity to have a "mens rea" or guilty mind. Yet it is a practical verdict giving the Home Secretary control of the insane criminal for an indefinite period.

Incidence of Mental Disorders Among Criminals.— The incidence of significant mental abnormalities among criminals depends, of course, upon our concept of what is a *significant* abnormality. There are the more radical criminologists who hold that most criminals are mentally abnormal. If they mean merely that most criminals are seriously maladjusted individuals, we would have to agree. Some years ago, Goddard and other enthusiastic early testers of intelli-

gence held that feeblemindedness was one of the great causes
of criminality. Time has not confirmed this contention. The
incidence of feeblemindedness among criminals is little more
than that of the general population.

In Massachusetts, where most defendants in the Criminal
Courts are given routine psychiatric examinations, the inci-
dence of frank psychoses is low—approximately 2 per cent.
When one includes the grossly feebleminded and the indi-
viduals with seriously warped personalities—the psychopaths
—among those with significant mental abnormalities, the per-
centage rises to 19.

Every type of insanity may have as a symptom the com-
mission of a criminal act, and the variety of psychiatric cases
that comes into the Criminal Courts is amazing. The dispo-
sition of the *frankly psychotic* is relatively simple; they are
committed to mental hospitals. The *psychopathic* and the
defective delinquents, on the other hand, frequently offer
baffling problems. They are neither insane nor normal.
Several of the European countries consider these individuals
as only partially responsible, and are indulgently lenient
toward them. Nebraska considers mental abnormality, short
of irresponsibility, as a mitigating circumstance.

From the point of view of society's protection, this is a
stupid policy. These conditions are constant and persistent
and they do not yield to our present methods of treatment.
The recently enacted Psychopathic Sexual Criminal Law of
Illinois is far more logical. It provides for a wholly inde-
terminate commitment of persons indicted for crimes when
they are "suffering from a mental disorder and not insane or
feebleminded, which mental disease has existed for a period
of not less than one year . . . coupled with criminal pro-
pensities for the commission of sex offenses." Psychiatrists
are too often thought of as sentimentalists. Many times we
find that we, rather than the Judges, are advocates of severe
sentences. The writer recalls urging a County Judge to give
a life sentence to a legally responsible arsonist who had
destroyed more than a million dollars' worth of property
during a period of four years. But the Judge decided that
ten years was long enough!

Castration has been used in certain types of chronic sexual

offenders in Europe for some years. It has recently been tried in this country with rather promising results. Scientifically, it is a far more logical procedure than the sterilization of sexual offenders and other criminals, which has been legalized in several states.

INCOMPETENCY PROCEEDINGS

In this country, there is a striking lack of unanimity in the type of procedure employed in the appointment of guardians of individuals who are incapable of administering their own affairs. There is even no agreement in which type of Court these proceedings should be instituted.

In twenty-nine states, the hearings are before a Judge; in eleven states, the issue is decided by a Jury; in five, there is a choice of Judge or Jury, and in three, the issue is decided by a commission. Apparently, it is generally held by legal authorities that the appointment of a committee to administer an individual's affairs is of far less importance than the appointment of a guardian for his person and his commitment to an institution. In these proceedings, fewer states require the participation of physicians than in commitment proceedings.

In Kansas, incompetency is passed upon by a Jury of six persons. The law requires that one of the six be a physician. In Massachusetts, the Court may require medical testimony or may require the alleged incompetent to be examined by Court appointed experts. South Carolina requires at least two practicing physicians as witnesses in incompetency proceedings.

COMMITMENT PROCEEDINGS

Commitment proceedings are heard by a Judge in twenty states, by a Jury in five states, by either a Judge or a Jury in nine states, and by a commission in nine states. There are two states in which the choice lies between the Court and a commission, and three states in which the choice lies between a Jury and a commission. In six of the fourteen states in which the issue of commitment may be passed upon by a commission, a Judge is the presiding member of the commission. Most of these commissions have physicians as members. In

the writer's opinion, the most satisfactory agency for determining the committability of an individual is a *permanently organized* commission on which there are both legal and psychiatric representatives.

There are only seven states in which the law does not specifically state that there must be *competent medical testimony* during the commitment proceedings. In recent years there has been widespread recognition of the necessity of getting insane individuals into hospitals quickly. Acute psychoses and acute exacerbations in chronic psychoses are frequent occurrences. Quick action is often necessary in order to preserve the life of the patient, and sometimes of the individuals around him. In many states it is still necessary to harbor the patient temporarily in the county jail. There are now more than twenty states that have statutes authorizing the emergency certification of insane patients, and then the subsequent determination, by Habeas Corpus proceedings, of the necessity of their being kept in a mental hospital. This is obviously a highly desirable method of procedure. Without some expeditious method of forcing insane persons to enter a hospital, serious harm may come to the mentally sick individual.

In the minds of the legal profession, there is great fear of the abuse of this privilege of summarily sending patients to a mental hospital. In the writer's opinion this fear is greatly exaggerated. There must, of course, be effective safeguards providing for immediate examination of the allegedly insane individual, and a legal hearing immediately following the examination, if such be demanded. Further, the allegedly insane individual has always the right of instituting suit against the persons who may be responsible for his unwarranted restraint; and the state medical licensing agencies have the right of taking away the license of a physician who lends himself to illegitimate commitment proceedings.

In those states in which individuals can be forced to go to mental hospitals after medical certification, the laws specify that *actual examination* of the patient must have been made; a mere history obtained from others is insufficient. Many of our larger communities have in their Health or Welfare Departments committing physicians, men experienced in psy-

chiatry, who at the request of some other physician or the police, will examine persons suspected of insanity to determine whether they are certifiable. Such a system greatly reduces the possibility of error.

MENTAL DISORDERS FOLLOWING INJURY

Post-traumatic Neuroses.—The number of suits following personal injuries that are annually brought in the Courts of the United States is amazingly great. This state of affairs is peculiar to our country. No doubt it can in part be explained by the fact that in our democracy there is an aggressive disgruntlement against the wealthy on the part of those economically less fortunate. There is the general feeling that corporations that are owned by the rich should lavishly compensate those who are injured, particularly if they be poor. This condition does not exist in our great sister democracy, despite the fact that English law forms the structural basis for the laws of all of our states but Louisiana. In all probability this is in part due to the fact that there has been a more passive acceptance in England of class and financial inequalities.

The writer vividly recalls an illustrative incident which occurred a few years ago. He was asked to go to a neighboring room in a London hotel where he was staying. There he found the corpse of an elderly man slumped over in a bathtub overflowing with scalding hot water. A few minutes later the hotel physician arrived and promptly made out a certificate in which the primary cause of death was "scalding." The writer suggested that in piecing the events together it seemed more probable that the man had died from acute cardiac failure or a cerebrovascular accident, and had been unable to turn off the water. He remarked to the hotel physician that, in America, a man in his position would hesitate to give scalding as a cause of death in fear of a lawsuit. Our English medical colleague promptly replied: "English people are not like Americans. They would never bring suit for anything like that!"

The realization on the part of the public that there is a great likelihood of recovering damages for almost any injury is no doubt an important factor, not only in the number of

malingerers who bring suit in our Courts, but an important etiologic factor in the high incidence of traumatic neuroses. A neurosis has as its basis a real unconscious purpose. Sometimes this is the gaining of sympathy; sometimes it is a method of flight from an undesirable situation; or it may be the acquisitive desire of an individual who feels that he has been unfairly deprived in the past of his rightful share of the world's goods.

The writer recalls that his first impression of a personal injury trial was its striking resemblance to an athletic contest. There seemed to be two teams with star performers, aided by expert coaches. The teams were carefully drilled. There were numerous trick plays and very complicated rules. The umpire was only calling the most flagrant fouls. It was hard to get the feeling that here was an extremely complicated medical problem that was being carefully investigated and presented, and would be decided on its scientific merits. Ten years' work in the Courts has not entirely erased this first disquieting impression.

The writer feels that there must be far-reaching reforms in our Court procedures if the number of personal injury suits brought is to be decreased. Some have advocated that the lay jury should be replaced by a jury of medical experts if a just evaluation of the seriousness of the injury is to be arrived at. The writer believes that a reform of our present system should at least provide for the testimony of *neutral experts* testifying on behalf of the Court. He feels that not only will the number of suits be decreased, but the number of traumatic neuroses will thereby be decreased.

In real traumatic neuroses, the *personality make-up* of the injured is of even greater importance than the *type of injury* which has been sustained. There are certain stable individuals who would be unlikely to develop a neurosis under any conditions. There are others in whom a neurosis follows the slightest injury.

There are probably few medical conditions whose development and outcome are more dependent upon the character of the immediate treatment than the neurosis following injury. Most individuals are in an unusually *suggestible* state following an injury. The attending physician, by careful treatment

and great reassurance, can do much to prevent the development of a neurosis. On the other hand, the suggestion of serious sequelae, lawsuits and so on by the physician, members of the family, or an ambulance-chasing lawyer, is frequently enough to sow the seed for the development of a neurosis.

The *promptness* with which the trial is held is of great importance to the post-traumatic neurotic. If the trial is postponed for a long period, the symptoms persist, and the longer they persist, the more difficult is their final eradication. Furthermore, it is greatly to the advantage of the patient to have the suit finally disposed of at time of trial by a lump sum settlement, rather than to have the outcome remain indefinite.

Few medicolegal problems are so complicated as the traumatic neurosis. The separation of the malingerer from the neurotic is often extremely difficult. Important work is being done by Dr. Helen Flanders Dunbar at the Presbyterian Hospital in New York City, which leads to the conclusion that there is a not inconsiderable number of individuals who are constantly being injured because of an unconscious drive toward self-punishment.

Post-traumatic Psychoses.—In an early paper Adolf Meyer gave the following *classification* of the post-traumatic psychoses which, in the writer's opinion, has not been improved upon:

1. Direct post-traumatic deliria.
2. Post-traumatic constitution (5 types: (a) with mere facilitation of reaction to alcohol and acute toxic or infectious processes; (b) with vasomotor neurosis; (c) with explosive diathesis [outbreaks of extreme irritability of temper]; (d) with hysteroid or epileptoid episodes; (e) with paranoid development).
3. Traumatic defect conditions (3 types: (a) primary defects allied to aphasia; (b) secondary deterioration with epilepsy; (c) terminal deterioration from progressive alterations of the injured parts).
4. Psychoses to which trauma merely contributes (2 types: (a) general paralysis; (b) manic-depressive and other transitory psychoses).
5. Psychoses from injury not directly affecting the head.

It is important to call attention to the symptom complex which has been described by Martland in prize-fighters which he calls the "punch drunk" condition. This generally results from repeated cerebral concussions and is associated with minute hemorrhages of the brain.

Although medical opinion is by no means unanimous, Courts have everywhere held that *general paresis* and *paralysis agitans,* occurring soon after injury, may be considered to have resulted from a head injury.

The role of *head injury* in criminal behavior is also a problem about which there is considerable disagreement. The writer believes that as a rule there is no relationship between criminality and head injury. On the other hand, he holds with Healy, Kasanin, et al., that severe head injury in childhood may lead to profound characterological changes which are often permanent. This is analogous to the effects of epidemic encephalitis. When this malady occurs in childhood, it frequently results in serious, persistent psychopathic behavior. On the other hand, when the affection occurs in adulthood, sequelae are neurologic rather than characterologic.

It is quite generally held that tolerance for alcohol decreases after a head injury. In his work with criminals, the writer has been impressed with the strikingly abnormal behavior sometimes displayed by drunken individuals who have had severe head injuries. In his experience, serious impulsive acts such as motiveless shooting are more likely to occur in intoxicated individuals with a history of head injury than in those in whom there is no such history.

COMPETENCE OF INDIVIDUALS TO MAKE CONTRACTS

The fact that a person is *psychopathologically* insane does not necessarily mean that he is *legally* insane. Courts have held that the person must be so incapacitated that he is unable to understand the nature and general effect of the transaction in which he enters, to be incapable of making a valid deed and contract. It has been generally held, however, that if the individual is under guardianship, contracts which he has made are automatically held void without inquiry. Some decisions have held that contracts and conveyances of insane persons are only voidable by the insane person.

Since the common-law conception of *marriage* includes consent as an essential element, the marrying of insane individuals is generally prohibited. There are statutes in eight states which make such a marriage automatically void. In twenty-eight states, such marriages are voidable. There are,

however, six states which permit marriage when the woman has passed her child-bearing period, which is legally held to be forty-five years. Nebraska, New Hampshire and South Dakota permit the marriage of an insane individual after that person has been sterilized.

There are now twenty-one states that grant *divorce* when one of the parties develops insanity subsequent to marriage. In all but one state insanity must have existed for a period of from two to ten years. There are eight states in which the insanity must have existed for a period of at least five years. About half the statutes specify that the existence of insanity is not sufficient, but that the patient must have been confined in a mental hospital during this period.

The contractual act of *writing a will* has a very important and special place in the law. A New Jersey Court held in 1820 that a person might be able to make a will and still not be able to make a contract because "most men have been cogitating over a will for some time and therefore require less cogitation than in comprehending a new business deal." This has been generally followed in this country as well as in England. Justice Bushrod Washington of the Circuit Court of the United States wrote a controlling opinion in the early part of the nineteenth century in which he held that even though an individual be in some degree senile, he was still competent to make a will. In this opinion, he said:

He may not be able at all times to recollect the names, the persons or the families of those with whom he had been intimately acquainted; he may at times ask idle questions, and repeat those which had before been asked and answered—and yet he might be entirely competent to make a will.

English Courts have held that the Testator does not have to be of perfectly sound mind, and that a delusion which is not of a kind to affect the will does not invalidate it. This has also been followed in America, and a Georgia statute specifically provides that an insane individual can, *during lucid moments*, make a will.

This liberal attitude in regard to the capacity of an individual to make a will is very practical and realistic. Many persons do not make wills until they are quite aged. However, the tendency of the Courts to consider the judgment of

an individual with delusions to be unaffected on issues on which his delusions do not bear, is medically unsound. The law conceives of the mind as made up of a large number of isolated compartments which are completely independent of each other. Scientifically, there is abundant evidence to show that mentation is a function of the entire brain and cannot be divided up into discrete elements.

There are forty states in which statutes deny the insane the right to vote, and thirty-six states have statutes disqualifying insane individuals from acting as jurors. Twenty-nine states have statutes providing that coitus with an insane or mentally defective woman, other than one's wife, is a felony. In several states this is considered rape. In eight statutes it is specifically stated that to be guilty of rape, the individual must have known that his partner was insane or mentally defective. There are two states that provide a similar penalty for women having sex relations with insane or mentally defective men.

The question of the *sterilization* of the mentally defective and the insane is one of great importance. The legality of sterilization of the defective cannot be questioned since the famous decision of the late Justice Oliver Wendell Holmes, in which he decided in 1927 in the case of Buck vs. Bell that "Three generations of imbeciles are enough." There are now eighteen valid statutes providing for the sterilization of the feebleminded, seven providing for the sterilization of the insane, and thirty-two providing for the sterilization of epileptics.

The Committee of the American Neurological Association for the Investigation of Eugenical Sterilization, in its 1936 report had no hesitation in recommending sterilization of the feebleminded. They felt that in cases of dementia praecox, the question was not a pressing one since the sexual urge is low and marriage and birth rate correspondingly low. The Committee felt that great caution should be taken in the sterilization of so-called schizoid personalities and people suffering from manic-depressive insanity. It felt that sterilization of epileptics should be adjusted on the grounds of the social situation rather than on its biology. It was felt that if the epileptic attacks were infrequent and the qualities of

the patient's personality were intact, sterilization should not be recommended.   .

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THE MEDICOLEGAL ASPECTS OF ROENTGENOLOGY

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The medicolegal aspects of roentgenology may be considered from three angles: (1) the presentation in court of testimony based on roentgenologic findings; (2) the preparation of the proper foundation for such testimony by accurate roentgen technic and correct interpretation of the films obtained; and (3) the legal responsibility of the individual employing x-ray apparatus for diagnostic or therapeutic purposes, or both.

TESTIMONY BASED ON x-RAY FINDINGS

The courts of the various jurisdictions throughout the country have established *rules* which govern the admission of testimony based on the interpretation of x-ray films. There is, as to be expected under these circumstances, no uniformity of these rules; therefore, a witness should inform himself of the rules *of his own jurisdiction* before he attempts to testify.

Jurors Should Not Interpret x-Rays.—The courts universally require that the films which form the basis of testimony should be a fair and accurate presentation of the parts examined. Most of the courts require that the films be exhibited and explained to the jurors by an expert, and many jurisdictions allow the jurors to examine the films and place their own interpretation upon them. A few jurisdictions do not require that the films actually be exhibited to the jurors, but they—the courts—are willing to accept the conclusions of the expert based upon his interpretation of the films. He must, of course, identify the films in court, where they may

be examined and interpreted by other experts if it is deemed
expedient.

It is obvious that the procedure which permits the jurors
to examine the films is at fault, because no group of laymen
can hope to have the proper appreciation of and give the
correct interpretation to the shadows which appear upon an
x-ray film. x-Ray films are not pictures but shadowgraphs
which can be properly interpreted only by one who has de-
voted years of study and practice to that particular branch
of the practice of medicine. If jurors, who are totally lacking
in that knowledge and experience, are permitted to survey
films, they will inevitably form the wrong conclusions in most
cases, thereby working a great injustice. Every effort should
therefore be made by those interested in this phase of medico-
legal testimony to have the courts in their jurisdictions adopt
a proper procedure for the presentation of roentgenologic
testimony.

Admission of x-Ray Films as Evidence.—Some courts
apply stringent regulations regarding the admission of x-ray
films as evidence and will permit the introduction of films by
the *plaintiff* only. Courts of a few jurisdictions require that
before the witness is permitted to submit films as evidence he
shall have either been present at the time or shall actually
have made the x-ray examination himself. Other courts are
more liberal; some will permit the introduction of films of a
normal individual for purposes of comparison with those
which form the basis of the action, and others have actually
ordered x-ray examinations, but only in cases when it was
deemed likely that such examinations would provide addi-
tional information.

Proper Identification of Films.—It will be noted that
all courts require the proper identification of any and all
films which form the basis of testimony. Films cannot be so
identified unless they are *properly marked* at the time the
examination is made, and since any x-ray film may become a
part of the evidence in some future litigation, it follows that
any and all films should receive the proper identification
marks at the time of the examination. Films should be stored
and filed *under the supervision of the person who conducts
the examination,* for the period of the statutes of limitation;

this is for the protection of both the patient and the doctor. The doctor who makes the examination has the right to insist —and he should insist—that he retain possession of the films. The courts have ruled that he is the legal owner of the films, because they are the part and parcel of the examination which forms the basis of his opinion.

The information obtained by an *x*-ray examination should be regarded as *privileged communication* between the patient and the doctor, and the latter should be careful not to divulge any of this information to anyone without the consent of the patient, unless ordered by the court.

NEED FOR PROPER PREPARATION AND CORRECT INTERPRETATION OF FILMS

It is well for the witness when giving testimony based on *x*-ray findings to remember the limitations of roentgenology. If the evidence is *clear-cut,* then definite statements should be made; if it is *equivocal,* the witness should frankly say so; and if, as sometimes happens, the *x*-ray findings shed no light on the problem, then again a frank admission should be made. Correct testimony is based on accurate diagnosis, which in turn is achieved by the proper correlation of the essential clinical data and the *x*-day findings.

If roentgenologic testimony is to be effective, it must be substantiated by unimpeachable interpretation of the films. No attempts should be made either, on the one hand, to *overemphasize* the roentgenologic aspects of the case, or, on the other, to make the roentgen findings *conform* to the clinical data if they are in disagreement. If the witness is successfully to defend this testimony in court, the films on which he bases his opinion must be a fair and accurate presentation of the part examined; correct diagnosis cannot be obtained from faulty films. If he is to produce films of the quality specified, then he must have a thorough knowledge of the standard *x*-ray technic and the ability to apply the same.

Standard Technic of Preparing *x*-Rays for Court Purposes.—This standard *x*-ray technic has been evolved through years of experience and is designed to produce the very results required by the courts.

Clear Demonstration of Lesion Essential.—The prime

requisite of correct x-ray technic is the clear demonstration of the lesion on the x-ray film, and the two important factors in obtaining this result are: (1) *correct positioning* of the patient in relation to the x-ray films (Fig. 36 *A* and *B*); and (2)

Fig. 36.—*A*, Faulty positioning of patient causes false appearance of dislocation. *B*, Film of the same patient made in correct position; no abnormality demonstrated.

the employment of sufficient technical skill to produce films of satisfactory *photographic value.* The proper observance of these rules of technic will ensure satisfactory results; yet it is surprising how often they are ignored, as is indicated by

Fig. 37.—*A*, Lateral projection shows normal appearing os calcis. *B*, Same patient. *Perpendicular projection demonstrates fracture.*

the frequency with which utterly worthless films are offered in evidence. Surely such films are not a fair exposition of the parts examined, hence they cannot form the proper foundation for correct testimony.

Incomplete Examination in Fractures.—Mistakes of omission are perhaps as common as those of commission in the x-ray diagnosis of fractures because the examination is incomplete. All too often the examination is terminated after one or two films are made and no further efforts to exhibit the fracture are undertaken (Fig. 37 *A* and *B*). Many fractures may be demonstrated by such simple methods, but others may be so situated in the body as to exhaust all of the resources of the examiner before they are detected (Fig. 38 *A* and *B*).
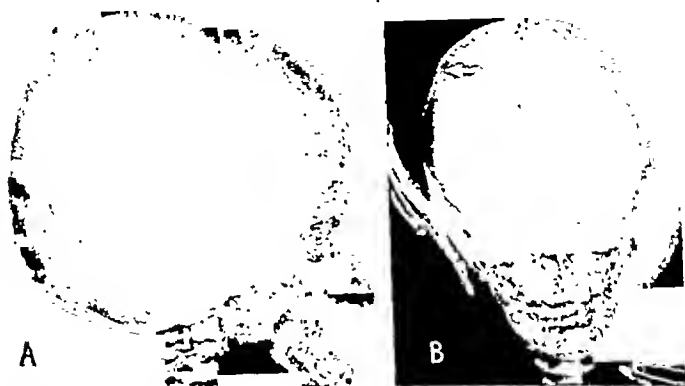

Fig. 38.—*A*, Films should be made from all projections when searching for fracture of skull. Lateral film fails to demonstrate fracture. *B*, Same patient. Anteroposterior film shows depressed fracture.

Special and ingenious methods of examination have been devised to cope with such situations; the examiner should be familiar with these refinements of x-ray technic if he is to demonstrate the fracture successfully (Fig. 39).

Examination Should Be Made Before Application of Dressings or Appliances.—The fundamental principle in the treatment of fractures is, of course, correct diagnosis, and one of the chief means of establishing the diagnosis is the x-ray examination; but the examination must be made before the application of surgical dressings or appliances or it may fail of its purpose. If opaque or semi-opaque dressings are applied to the affected part preceding the x-ray examination, then a confused image will appear on the films and accurate diagnosis will be impossible.

After the fracture has been reduced films should be made to provide a permanent record showing that a *satisfactory anatomic result* has been obtained. The x-ray films form a very important part of the record in the treatment of fractures, because they usually provide graphic evidence of the correct diagnosis and of the success or failure of the treatment. The courts recognize the importance of the x-ray examination in the treament of fractures and they have

Fig. 39.   Fig. 40

Fig. 39.—Special technic used to demonstrate *inaccessible parts* of the body. Semilateral view of upper thoracic vertebrae. Direct lateral view cannot be obtained.

Fig. 40.—Part of transverse processes developing as apophyses, presenting appearance of fractures. No history of injury.

repeatedly held that the *failure to check results* by means of the x-rays constituted negligence on the part of the attending physician, for which he was liable.

Diagnosis of Fracture Should Not Depend on Fluoroscopic Examination.—x-Ray diagnosis of a fracture should not depend upon fluoroscopic examination alone, because this method of examination is *too* unreliable. It is true that fractures in which the fragments are definitely separated may be demonstrated by this method, but fluoroscopic examination

may not disclose the full extent of the injuries. More than one fracture may be present, one producing deformity and the other not. The first fracture will easily be detected by the fluoroscope while the other may be overlooked. Then, too, impacted or incomplete fractures or epiphyseal separations with no initial deformity may be extremely difficult to demonstrate on the fluoroscopic screen by even the most skilful examiner. Failure to recognize any one of these conditions may place the attending physician in an embarrassing position. For instance, if he does not realize the presence of an impacted Colles fracture, and hence does not institute proper treatment, the end result may be a painful and deformed wrist joint which is either partially or wholly immobile. The patient is likely to seek redress under such circumstances.

Pitfalls in the Diagnosis of Fracture.—Certain pitfalls in the diagnosis of fracture deserve consideration, because they may be of medicolegal importance. A comprehensive knowledge of the *normal* growth and development of the osseous system, its appearance at all ages in life and the manner in which some disease processes affect it, is absolutely essential in the diagnosis of fractures. Normal epiphyses may sometimes appear on x-ray films in the guise of fractures and abnormal or extra epiphyses will certainly present diagnostic problems.

Lesions of the Spine.—All of these factors enter into the diagnosis of lesions of the spine, which is the subject of many roentgenologic controversies. In the first place, it is difficult to depict the spine clearly and accurately on an x-ray film because of the irregularity of its shape and the complexity of the anatomic relations of the vertebrae. The shadows of adjacent vertebrae overlap one another on the film in such a manner that it is difficult to differentiate them. Moreover, the picture may be further complicated by the superimposition of the shadows of the thoracic and abdominal viscera; and then, to make the picture more confusing, the spine frequently is the site of congenital abnormalities. When these anatomic abnormalities are gross and are quite apparent, no question of fracture arises, but they do cause definite deformities of the spine which may have medicolegal significance.

Some of the *minor abnormalities of the spine* may also
be of medicolegal importance. In the lumbar region the trans-
verse processes of the first, and less frequently of the second
and third vertebrae may develop as apophyses and never
unite with the main body of the bone. When the transverse
processes do develop in such a manner they present a picture
not unlike that of a fracture (Fig. 40). The inferior articulat-
ing facets of the lumbar vertebrae may develop in the same

Fig. 41

Fig. 42

Fig. 41.—Extra centers of ossification for articulating processes, simulating
fracture.

Fig. 42.—Loose fragment in knee joint due to osteitis desiccans and not fracture.

manner and produce the same diagnostic problems (Fig. 41).
Extensive damage may be done to the spine without produc-
ing any fracture, the injuries being confined to the ligaments
and intervertebral disks. Very few if any roentgen changes
are produced in these cases because the injured parts are not
demonstrable upon an *x*-ray film. For this reason the method
of diagnosing dislocations of the *intervertebral disks* by the
introduction of contrast media into the spinal canal has been

accepted with great enthusiasm.   The results of this method have been satisfactory in the hands of experienced observers, but the technic is complicated and must be applied exactly. The examination will be doomed to failure unless the technic employed is absolutely accurate; faulty or careless technic will inevitably lead to false and erroneous conclusions.

Bony Changes Simulating Fracture.—Certain disease processes which affect the osseous system cause destruction and

Fig. 43   Fig. 44

Fig. 43.—Deformity of hip joint caused by healed Perthes' disease, not a fracture.

Fig. 44.—Pathologic fracture of femur. Note area of destruction in shaft.

fragmentation of bones and produce changes on an *x*-ray film which simulate fractures. *Osteitis desiccans* (Fig. 42) and *aseptic necrosis* (Fig. 43) are two of the chief offenders in this connection; they produce lesions in or near the various joints of the body, in the hands or feet, or in the spine, which are great masqueraders of fractures. Many disease processes such as *hyperparathyroidism* and *osteomalacia* as well as primary (Fig. 44) and metastatic (Fig. 45) *neoplasms* cause widely disseminated lesions of the osseous system which often

Fig. 45   Fig. 46

Fig. 45.—Pathologic fracture of neck of humerus. Many foci of bone destruction seen in head of humerus.

Fig. 46.—The small fragment of bone posterior to astragalus is a sesamoid bone, not a fragment of the astragalus.

Fig. 47.—*A*, The sesamoid bone under the great toe often appears, in the anteroposterior view, to be fractured. *B*, Lateral view shows smooth edges of bone. No history of injury.

result in pathologic fractures or collapse of the bodies of the vertebrae. These are but a few of the conditions which must

be taken into consideration when making a roentgenologic diagnosis of fracture.

Sesamoid Bones Simulating Fracture.—Some of the sesamoid bones which appear so frequently in the region of the joints may also lead to an erroneous diagnosis of fracture; a good example of this is the *os trigonum* which sometimes appears on the posterior aspect of the astragalus (Fig. 46). Occasionally these sesamoid bones remain segmented and ap-

Fig. 48.—Tripartite patella, not a fracture. These anomalies are usually symmetrically bilateral.

pear to be fractured (Fig. 47), but close examination will usually establish the correct diagnosis. The patella (Fig. 48) and the sesamoid bone beneath the distal end of the first metatarsal bone are the best representatives of this condition. It is often necessary to take films of the corresponding joint of the opposite extremity in order to clarify this diagnostic problem.

x-Rays of the Chest.—The *x*-ray film, because it is such an essential feature of any examination of the chest, plays

an important part in the settlement of the many medicolegal disputes relative to chest lesions. The medicolegal aspect of the case may develop quite unexpectedly and may not be apparent at the time of the *x*-ray examination; therefore, if it is at all expedient, *x*-ray films of the chest should be kept *indefinitely* and not merely for the period of the statute of limitations.

If a film of the chest is to have any value as the basis of medicolegal testimony, it must be made at a *film-target distance* of not less than 48 inches and the *length of exposure* should not exceed 0.1 sec. (Fig. 49). These criteria are
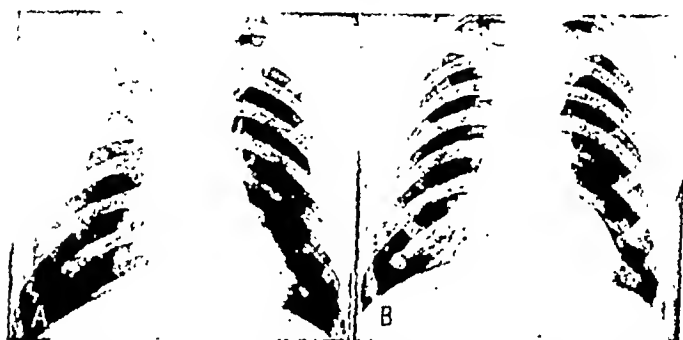

Fig. 49.—*A*, Clouding of right upper lung field due to slight rotation of shoulders. Demonstration of effects of faulty technic on chest films. *B*, Same patient in correct position. Lungs are clear.

established to ensure clear definition of the thoracic organs on the film, because if the film-target distance is less than 48 inches too much distortion will be produced; if the exposure time exceeds 0.1 sec., transmitted motion from the heart will cause blurring of the image.

The study of some intrathoracic lesions requires supplementary *fluoroscopic* examinations, *stereoscopic* examinations, or films made from different projections as well as on both inspiration and expiration. This is particularly true when studying the heart and great vessels.

Limitations of Roentgenology in Pulmonary Lesions.— The witness, if he is wise, will remember the limitations of roentgenology when he is testifying regarding pulmonary lesions, because he will realize that oftentimes pulmonary con-

ditions change their roentgen appearance very rapidly, necessitating a complete revision of the conception of their etiology
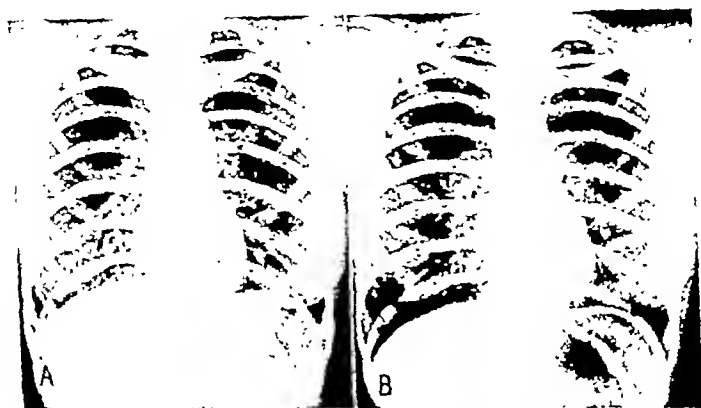

Fig. 50.—*A*, Localized area of infiltration in left upper lobe suggesting an early tuberculous lesion. *B*, Rapid clearing of lesion within two weeks establishes diagnosis of pneumonia.

(Fig. 50). Many different pulmonary lesions produce similar or identical roentgen appearances which renders impossible
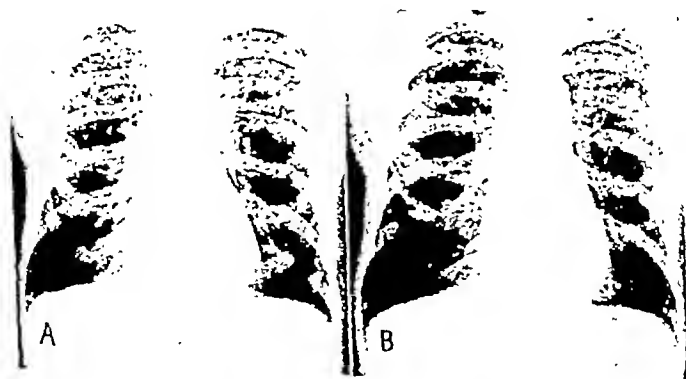

Fig. 51.—*A*, Extensive changes in each upper lobe, due to bronchopneumonia and not tuberculosis. *B*, Lungs have cleared in two weeks.

an objective diagnosis from a single examination (Fig. 51). Some cases will require *repeated* examinations at intervals over a period of time before the correct diagnosis is established.

Silicosis.—Silicosis has become the storm center of many
hotly contested medicolegal controversies since the enactment
of laws which make industrial diseases compensable. A fair
estimate of the importance of this subject may be gained by
a review of the voluminous literature pertaining to it. Since
the *x*-ray films form such an important part in the diagnosis
of silicosis, much of the literature is devoted to the roentgen
appearance of the disease, and careful investigators have
established definite diagnostic criteria, which, if properly ap-
plied, will lead to the correct diagnosis in many cases. The
differential roentgen diagnosis of silicosis is not easy, how-
ever, because the *x*-ray findings are so often imitated by
many other diseases. For the very reason that the roentgen
findings are so important in the diagnosis of this disease it
is incumbent upon the observer to be very careful in the
formation of his opinions and in the presentation of testimony
based upon them. He should avoid making dogmatic state-
ments unless absolutely sure of his ground, always remem-
bering that it is often impossible to make a positive roentgen
diagnosis of silicosis in a given case. Because decisions in-
volving cases of silicosis are among the most important in
the medicolegal domain, and because the diagnosis of this
disease presents so many problems, anyone who must make
these decisions and solve the diagnostic problems must have
a comprehensive knowledge of the subject.

LEGAL RESPONSIBILITIES OF THOSE USING *x*-RAY EQUIPMENT

Need for Records of Total Dosage in Every Case.—
The courts have established certain legal responsibilities for
anyone employing *x*-ray apparatus either for diagnostic or
therapeutic purposes. The roentgenologist must have an exact
knowledge of the *output* of each piece of *x*-ray apparatus at
his command. If he is engaged in *x*-ray therapy he should
predetermine the total dosage which may be safely applied in
each case and he should be careful not to exceed the pre-
scribed amount. He should keep accurate records of the
number and *intensity* of the treatments which are given the
patient in order that he may have, at any time during the
treatment, a precise knowledge of the amount of irradiation

that the patient has received. If he is called upon to defend his position in court, he must be able to prove that he has had his x-ray apparatus calibrated, he must display a sufficient knowledge of physics to show that he has calculated the dosage correctly, and he must establish by his knowledge of pathology and biophysics that he has employed the correct dosage for the lesion which he has treated.

High voltage, or *deep x-ray* therapy may cause unavoidable changes in the skin and underlying structures, and it may sometimes produce an impairment or complete cessation of some of the physiologic functions of the body. If such changes are anticipated the patient should be *warned* before the treatments are instituted.

Cumulative Effects of Irradiation.—Just as much care should be exercised when using superficial x-ray therapy as is observed when the higher voltages are employed. The chief danger in this form of therapy arises from the fact that small amounts of irradiation are applied at any single treatment, which produces a false sense of security in the mind of the therapist. If he does not realize that the effects of irradiation are cumulative and if he repeats the treatment too frequently, he will cause severe skin damage. Such a sequence of events has been the cause of much litigation.

Too Frequent or Prolonged Exposures.—All x-ray apparatus used for diagnostic purposes should be calibrated so that the examiner may know the *exact amount* of irradiation which is applied to the skin at each and every x-ray examination. This is to prevent damage as a result of too frequent or too prolonged exposures.

Dangers in Fluoroscope.—Special mention should be made of the fluoroscope, apparently a harmless piece of apparatus but really a very powerful instrument which is capable of producing a surprising amount of energy. It has, in fact, been the cause of more litigation in recent years than any other piece of x-ray apparatus, because the operator did not realize its dangerous potentialities. This is an extremely important matter, as many cases have been reported in which irreparable damage has been done to either the patient or the fluoroscopist as a result of *unduly prolonged* examinations. Most of these unfortunate instances have occurred in the

dangerous practice of *setting fractures* under the fluoroscope, in searching for *foreign bodies* or in pursuing *gastro-intestinal examinations*.

Electrical Shock.—Modern shock proof apparatus is rapidly replacing the older non-shockproof type, so that the danger from electrical shock is being eliminated. If non-shockproof equipment is used, the patient should receive clear and explicit instructions so that he may fully understand what is expected of him in each step of the examination and not subject himself to electrical shock.

Extreme caution should be observed when a *third person* is present in the examining room during an x-ray examination, because while the operator's attention is concentrated on the patient, this third person may so place himself in relation to the x-ray equipment as to receive an electrical shock.

All x-ray equipment should be carefully checked routinely, as pronounced changes in its output may occur quite suddenly without previous warning.

THE LEGAL ASPECTS OF OCCUPATIONAL DISEASES

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DURING the past decade the problem of occupational diseases has received nation-wide attention, because of the publicity given during this period to the existence of such disease hazards in various industries. This has served to emphasize the importance of the subject of industrial hygiene, and has prompted the enactment of legislation for the purpose of preventing occupational disease injuries and providing compensation therefor. In considering the legal aspects of this problem, it is essential to review briefly the developments that have resulted in the enactment of the compensation statutes.

Workmen's Compensation Acts.—The Workmen's Compensation Acts are of comparatively recent enactment, the first act held to be constitutional having been enacted in the year 1911. Since that time all of the States except Mississippi have enacted such laws. They resulted from the economic need to impose upon an employer the obligation of an insurer for the protection of his employees against accidental or traumatic injuries. Prior to their enactment, the sole remedy of an employee who sustained accidental injury was to sue his employer for damages at common law. This litigation eventually proved to be unsatisfactory for the determination of this type of dispute; its procedure was complex, the law permitted dilatory pleadings, resulting in extensive delays, while the cost of the litigation was exceedingly burdensome to the injured employee and to his employer. The compensation acts simplified procedure, expedited hearings, and enabled the injured employee to procure a speedy and inexpensive determination of his cause, with the prompt payment of compensation benefits. The compensation acts limited the employer's liability as to amount of benefits, but assured the

employee of his compensation, irrespective of the negligence of the employer or the employee.

Original Acts Covered Only Accidental or Traumatic Injuries.—The original Workmen's Compensation Acts were designed primarily to provide compensation for accidental or traumatic injuries, and not for occupational disease injuries. Accidents occur at specific times, at specific places, and there is usually no question as to the employer in whose employment the accident has been sustained. Occupational diseases, however, are often slow of development and may be unknown to the employer and to the employee until disability occurs. The injuries sustained are continuing injuries over an extended period of time, and may be incurred while the employee is working for one of several employers. Therefore, the legal problems presented by occupational disease injuries and the liability for such injuries that is imposed upon the employer differ materially from the problems presented by accidental injuries.

Status of Acts with Respect to Occupational Diseases.—The Workmen's Compensation Acts, when enacted, were not uniform in the language they employed as to the injuries proposed to be made compensable thereunder. All of them purported to provide compensation for injuries of traumatic origin, that is, injuries produced by wounds or bodily violence. Some of them specifically exclude occupational disease injuries. The decisions of Courts construing the nature of the injuries proposed to be compensated by these acts likewise were not uniform and, in some States (the minority) the Acts, in their original form, were construed to include occupational disease injuries, while in others (the majority) they were construed to exclude such injuries. In many of the States schedules for occupational disease injuries have been added by way of amendment to the original acts and, under these statutes, compensation is provided for such diseases in the same manner as compensation is provided for injuries occasioned by accidental means. In some of the States that deny compensation to an employee for occupational disease injuries under the compensation statutes, the Courts have held that the employee retained his common law right of action against his employer where the disease injury was caused by negligence; in other

States, this common law right of action on the part of the employee has been denied.

Legal Liability of Employer for Occupational Diseases.—The legal liability for occupational disease injuries has always been closely related to the legal liability of an employer for accidental injuries sustained by employees during the course of employment. Under the common law and also under the earlier European Codes, employers were liable to the employees only for injuries sustained by accidental means and caused by the employer's negligence. In many of our States, common law rights of action for occupational disease injuries are available to employees, recovery in such actions being dependent upon proof of the negligence of the employer, with the right in the employer to interpose his common law defenses.

These actions take the form of the *typical negligence suit,* wherein the employee seeks to recover damages for his alleged injuries. The plaintiff's declaration contains allegations that the defendant employer has caused the plaintiff to work exposed to alleged occupational disease hazards; that the defendant knew or should have known that certain materials used in the industrial process were harmful, dangerous, or destructive to life or health in the human body; that the defendant was guilty of negligence in failing to provide for the employee a safe place in which to work or safe appliances with which to work, and thereby harmful substances, consisting of dusts, fumes, or gases, became introduced into the plaintiff's body, causing him permanent injury.

The *defense pleadings* raise the defenses of assumption of risk by the plaintiff, the plaintiff's contributory negligence, the negligence of the plaintiff's fellow servants, and a general denial of the allegations of negligence as charged by the plaintiff. Upon trial, the plaintiff offers evidence as to the existence of the hazard in the working areas where he has been employed; of defective machinery, equipment and industrial processes; of the failure of the employer to furnish efficient protective equipment. Technical testimony is offered as to the nature of the industrial process and the defective equipment furnished by the employer. Medical testimony is then offered, describing the disease from which the plaintiff is

alleged to be suffering, its insidiousness, and the inability of the medical profession to effect its cure, with a prognosis of short life for the plaintiff.

The real bases of the defense are the failure of the plaintiff to prove the defendant's negligence, and the medical testimony that may be offered to contradict the testimony offered by the plaintiff.

Methods of Common Law Trial Unsatisfactory.—It is not necessary to emphasize the *seriousness* of this type of litigation. At the conclusion of the trial the case is submitted to a jury for its determination of certain questions of fact, including (1) whether or not the plaintiff has sustained occupational disease injury as alleged in the pleading; (2) whether or not the defendant employer has been guilty of negligence in causing the occupational disease injury complained of, and (3) the evaluation of the injury, if found to exist, in terms of a jury verdict. In most of these cases the testimony offered relating to the nature of the injury and the extent of disability is highly technical, the medical witnesses of either side controverting the testimony of the other, with the result that the jury may become confused as to the merit of the claim, and render a verdict that is frequently affected by the *personal prejudices* or *sympathies* of the respective jurors.

To both parties, this litigation means *substantial expense.* The employee usually retains counsel under an agreement to pay to him a large percentage of the amount recovered and, in the event of a plaintiff's verdict, after counsel fees and the expenses of the trial have been paid, he receives an inadequate proportion of the jury's award.

From the standpoint of the *employer,* his entire relationship with his employees may be seriously affected. In many instances, he may be subjected to suit by groups of his employees, many of the claims represented by these suits being baseless, but his only method of defense is to endure long, expensive and harassing trials in Courts of law, and the payment of jury verdicts.

The honest *employee* desires only reasonable compensation for the injuries that he has sustained during the course of his employment; on his part, the honest *employer* desires to provide reasonable compensation for those injuries. The

method of common law trial to determine these disputes has, therefore, proved unsatisfactory, and has created the need for some more suitable method. This has been found in the enactment of occupational disease compensation statutes, or the amendment of the workmen's compensation statutes to provide compensation for these injuries.

Occupational Disease Compensation Statutes.—At the present time, twenty-four States, the District of Columbia, Hawaii and Puerto Rico have enacted specific occupational disease compensation statutes, whereby compensation is provided in a manner similar to that in which compensation is provided for industrial accidents.*

The employee who becomes subject to these statutes surrenders his right of common law action against his employer for these injuries, and is limited to recovery of the amount of compensation *provided in the law*. The employer's common law defenses are abolished by the statute and upon him is imposed the liability of an insurer for the health of his employees against those diseases that are made compensable. His liability for these injuries is also limited to the amounts of compensation specified by the law.

The occupational disease compensation statutes that have been enacted by the several states are not uniform. They differ as to the diseases made compensable; they provide different methods of procedure for the determination of injury; the amounts of compensation payable vary; they prescribe various periods of limitations for the filing of claims and they require various terms of employment in order for the injury to be compensable. However, the following basic provisions are common to all of these acts:

Establishment of an Agency for the Administration of the Law.—The occupational disease compensation acts generally

* California, 1919	Maryland, 1939	North Carolina, 1935
Arkansas, 1939	Massachusetts, 1932	North Dakota, 1925
Connecticut, 1927	Michigan, 1937	Ohio, 1921
Delaware, 1937	Minnesota, 1927	Pennsylvania, 1937
Idaho, 1939	Missouri, 1931	Rhode Island, 1936
Illinois, 1936	Nebraska, 1935	Washington, 1937
Indiana, 1937	New Jersey, 1924	West Virginia, 1935
Kentucky, 1934	New York, 1920	Wisconsin, 1919

District of Columbia, 1928; Hawaii, 1935; Puerto Rico, 1935

provide for the administration of the law by the same Bureau or Commission that administers the Workmen's Compensation Act. Such agencies and the officers connected with them are usually familiar with the industrial conditions that would be involved in claims arising under the occupational disease law, and, by placing its administration under the existing Bureau or Commission, the cost of the establishment of a new agency may be avoided. The existing agency thereby retains jurisdiction over all disputes involved in the relationship of employer and employee, and the convenience of the parties is better served with respect to the filing of claims, hearings, medical examinations, supervision of state funds, and other administrative functions.

Method of Diagnosis of the Disease and Evaluation of Disability.—This duty is likewise most often assigned to the agency administering the Workmen's Compensation Act.

In many instances, for the purpose of determining the fact of injury and the resulting disability, provision is made in the law for medical advisors of medical boards to advise the administering agency as to the medical features of the claim. The litigants are still privileged to offer their own medical testimony, but in our large industrial centers, where most of these claims arise, the average medical practitioner is often not familiar with the intricacies of diseases that may be classed as occupational. In any event, the medical questions involved in these claims are always controversial and, by the use of medical examiners or boards, the administrative agency has the benefit of the advice of unprejudiced, impartial experts, trained in the diagnosis, care and treatment of such diseases, and better qualified to evaluate the existing disability.

Method of Determining the Diseases to Be Compensated.—To define the diseases to be made compensable, the statute may include either the general coverage method or the schedule system.

In those states having *general coverage* laws, the statutes purport to provide compensation for any and all "occupational disease injuries," which term may or may not be defined in the law. The purpose of such laws is to provide compensation for those diseases that are characteristic of and peculiar to

the particular occupation in which the employee is engaged and not those diseases that are common to everyday life. Considerable difficulty, however, is experienced in attempting to define "occupational disease," and the principal objection to general coverage statutes is based upon the apprehension that the administration of such laws will ultimately lead to general health insurance, the employer becoming liable for all the human ills to which his employees may be subject during the course of their employment.

The chief advantage of such laws lies in the fact that as new diseases develop with the use of new materials or processes in industry, employees contracting them may receive compensation, even though such diseases may not be known or named at the time the law becomes effective.

By the *schedule system*, which sets forth in the law by name those diseases for which compensation may be payable, the employer is apprised of the exact diseases for the compensation of which he becomes responsible, and may, therefore, be inclined to make every effort to protect his employees against the particular disease hazards that exist in the processes in which they are engaged. The sole objection to this method is that employees may sustain some disease injury not covered by the schedule.

Limitation Provisions.—These provisions relate either to: (1) the period of employment and exposure within the State as a pre-requisite for the allowance of the claim, or (2) the time within which claim must be filed after the injury.

The purpose of requiring a definite period of employment and exposure within the state is to avoid the imposition of legal liability upon an employer for injuries to employees that have been sustained in other states or other industries, but which, because of their gradual development, are not detected until some later time.

With respect to the *filing of claims*, all the Workmen's Compensation Acts provide some period of limitation after the injury within which claim must be filed, in order for the administrative agency to have jurisdiction. Occupational diseases differ from accidental injuries in that it is not always possible to place exactly the time and place of incidence of the disease and, in some states, the Courts have held that

the period of limitations runs from the date of last exposure to the hazard or the termination of employment, while in others the period of limitations runs from the date of discovery by the plaintiff that he is suffering from the disease. In order to accomplish an equitable administration of the law, and provide for the prompt treatment and cure of the disease injury, as well as its compensation, it is essential that some reasonable time limit be set within which claims must be filed.

Programs of Prevention.—Many state laws make provisions for the adoption of rules and regulations for the control and prevention of all types of personal injuries to employees. The administration of these rules and regulations may be assigned to the state administrative agency, the State Department of Health, or the State Department of Labor.

In thirty-one States there have been established *Bureaus of Industrial Hygiene* that are available for the purpose of assisting industry in conducting its business in a manner that will fully protect the health of employees. Their services include reporting systems for occupational disease injuries, inspection services, with trained technicians to determine the existence of potential hazards, and research departments to make surveys for the purpose of controlling or eliminating such hazards as may be found to exist.

The importance of programs of prevention cannot be overemphasized. Employees are more interested in the maintenance of their health than in compensation for injuries to that health, while to employers the *cost of prevention* of occupational disease injuries will prove to be but a small fraction of the *cost of compensation* therefor.

Liberalization of Benefits.—Since the enactment of the occupational disease compensation laws, many of their provisions have been amended and will continue to be amended as experience in administration develops the need for change in the laws. The recent tendency has been to liberalize compensation benefits and to broaden the general scope of the law. The hysteria that prevailed some years ago with respect to this legislation has been gradually allayed and the public generally has adopted a saner attitude with respect to this subject.

As more information has been developed about the problem of occupational diseases, there have been increasing evidences of cooperation of employers, employees, and Federal and State agencies to eliminate potential disease hazards in industrial operations, and it is evident that the occupational disease compensation laws now in effect have accomplished the purposes for which they were designed—to provide reasonable compensation for the injuries sustained and to provide the necessary machinery for the prevention of such injuries.

THE MEDICAL ASPECTS OF OCCUPATIONAL DISEASES

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An occupational disease may be defined as one "arising from and peculiar to a given occupation,"[1] or as one "which is due to causes and conditions characteristic of and peculiar to a particular trade, occupation, process or employment."[2] Either definition will serve as a basis for the discussion of the subject from a medical point of view, but it must be kept in mind that the term "occupational" has certain legal connotations in the twenty-four States in which disability resulting from one or more occupational diseases is compensable under workmen's compensation laws.

Historical.—Some of the effects of occupation on health have long been recognized. Hippocrates is said to have described a case of lead poisoning in a lead worker as well as one of dust disease in a miner. Pliny recorded the use of transparent bladders held before the face as a protection for men engaged in making oxides of lead. In 1700 Ramazzini published the first book devoted entirely to diseases of industrial origin.[3] The first author of the modern industrial age was C. Turner Thackrah, the English physician whose book on the influence of occupation on health was published in 1830. Since that time, and particularly in the last forty years, the literature on industrial medicine has grown to considerable size. A few of the volumes and journals are listed at the end of this paper.

The last twenty-five years have seen the rise of numerous agencies devoted to the prevention of occupational diseases. When the outbreak of World War I stopped the importation of many chemicals hitherto obtained from European countries, American industries undertook the manufacture of these mate-

rials and discovered for themselves the many hazards asso-
ciated with such manufacture. The simultaneous and
interrelated growths of the automobile, rubber and petroleum
industries produced many new risks to the health of employees
in those industries. Other potential hazards appeared in the
production of rayon and plastics. Public interest in the pre-
vention of occupational diseases grew to such as extent that,
when the Social Security Act was passed in 1935, funds were
made available for the establishment of industrial hygiene
units in many State Health Departments.

Causes of Occupational Diseases.—Any condition or
manner of working in an industrial environment which can
cause an occupational disease is called an "industrial hazard."
The following classification of industrial hazards has been
adapted from that of Dublin and Vane.[4]

1. *Skin Irritants:*
     (a) Mechanical: heat, cold, ultraviolet and x-ray radiation.
     (b) Plant: poison ivy, certain woods and grain dust.
     (c) Vital agents: bacteria, fungi and mites.
     (d) Chemical agents: inorganic, such as cement, alkalis and nickel; or
         organic, like tar, oils and turpentine.

The relative importance of industrial skin disease is evi-
dent from the report of the Statistical Department of the
Industrial Commission of Wisconsin, which states that out of
a total of 652 cases of occupational diseases compensated in
1938, the number of cases of occupational dermatitis was 439,
i.e., 67.3 per cent of the total. The statistics from many other
sources provide similar evidence.

2. *Poisons:*
     (a) Metals: lead, arsenic and mercury.
     (b) Organic compounds: coal tar group, *e.g.,* benzol and aniline; and
         the petroleum group, *e.g.,* carbon tetrachloride and methanol. Sev-
         eral hundred different chemicals with varying degrees of toxicity
         are included in this classification. New compounds are constantly
         being put in service, sometimes without any previous inquiry into
         their harmful potentialities.
     (c) Gases: the asphyxiants and the irritants. Examples of the former
         are carbon monoxide and carbon dioxide; of the latter, ammonia,
         sulfur dioxide and hydrogen sulfide.

3. *Dusts:*
     (a) Organic: flour, tobacco and grain, sometimes responsible for the
         origin of asthma and other manifestations of sensitivity.

    (b) Inorganic nonmetallic: silica (SiO_2), asbestos, coal, marble and cement. Silica is by far the most important member of this group because it is the cause of the pulmonary fibrosis known as "silicosis." Asbestos is a rare dust. The others, of themselves, seldom cause significant pulmonary injury. When mixed with silica they may modify the typical silicotic reaction.

4. *Abnormalities of Temperature and Humidity:*
    (a) Exposures to high temperatures, with or without an accompanying high humidity, may cause heat exhaustion, heat cramps or heat stroke.
    (b) Exposures to sudden variations of temperature tend to increase the incidence of pneumonia.[*]

5. *Abnormalities of Air Pressure:*
    Work under increased air pressure is the cause of caisson disease, as seen in divers and tunnel workers.

6. *Infections and Communicable Diseases:*
    The commoner examples are the oil furunculosis of machinists, anthrax among handlers of fresh hides, and tularemia in rabbit dressers.

7. *Radiant Energy:*
    (a) x-Rays and radium cause eczema in the absence of suitable protective measures.
    (b) Ultraviolet rays may give rise to conjunctivitis and corneal burns in improperly guarded welding operations.
    (c) The infrared rays from molten steel or glass are considered responsible for the formation of cataracts.

8. *Defective Illumination:*
    Too much or too little light or wrongly placed light causes fatigue and contributes to the impairment of vision.

9. *Repeated Motion, Pressure and Shock:*
    The results usually attributed to the use of tools actuated by compressed air are myositis, bursitis and synovitis. Telegrapher's cramp is included under this heading.

10. *Dampness:*
    This condition tends to increase predisposition to respiratory and neuralgic affections, especially at low temperatures.

Channels of Entry for Industrial Poisons.—Most cases of poisoning seen in general medical practice are due to the ingestion of a single large dose of some poisonous substance. In industrial practice the great majority of cases are due to the inhalation of multiple minute doses of the toxic material over a period of months or years. The difference between *absorption by ingestion* and *absorption by inhalation* might be illustrated as follows: To use the terminology of the traffic engineer, the gastro-intestinal tract is a "through street." Ingested material, especially in small amounts, may pass through the whole tract without being absorbed at all. Even if it is absorbed the minute dose of poison is compelled to pass through the liver and may be excreted in the bile. In

contrast, the respiratory tract is a "dead end" street. While some of the inhaled material is filtered out in the nasal passages and some is deposited on the walls of the pharynx, most of the remainder reaches the lungs. The only escape is by way of the expired air. That portion which is not exhaled is absorbed directly into the pulmonary circulation, a process comparable to intravenous injection.

Certain *physical factors* are of importance in connection with the inhalation of toxic substances. Except in the case of asbestos fibers, dust particles greater than 10 microns in any dimension do not reach the lung in significant amounts. The most dangerous particle size for dusts is from 1 to 3 microns.[6] The temperature of the workroom governs the rate of vaporization of such toxic solvents as benzol. The specific gravity of a gas or vapor determines whether that gas or vapor will rise to the upper part of the workroom or tend to collect in the lower parts near the breathing level of the workmen.

In addition to entry by inhalation and ingestion, it is well established that some few substances used in industry can penetrate the unbroken skin in quantities sufficient to produce toxic effects. Such substances are always good solvents of fats. The best example is benzol.

One other point with respect to absorption. Just as in medicine, a certain dose of a given drug is required to secure a therapeutic effect, so in occupational poisoning, there are *threshold limits* which must be exceeded before a toxic effect is produced. So long as silica is not present in inspired air in quantities greater than 4,000,000 particles per cubic foot (particle size less than 10 microns in greatest dimension), it is not likely that a disabling silicosis will develop during a working lifetime. Similarly, so long as the carbon monoxide concentration in the workroom air is kept below 100 parts per million, no ill effects are to be expected among persons exposed for eight hours or less.

Diagnosis.—In general it may be said that symptomatology is not a dependable guide in making a diagnosis of any occupational disease. The pain of lead colic may simulate the pain of intestinal obstruction or appendicitis. Dyspnea, which is a common complaint of patients with silicosis, is a part of the symptom complex of many other diseases.

The chief reliance in diagnosis is the *history of adequate exposure* to an industrial hazard. That exposure must be adequate both in time and severity. A workman does not acquire silicosis by working for a week in a very dusty granite cutting shop. The development of that disease requires years of exposure. On the other hand, hydrocyanic acid poisoning may follow an exposure of minutes to a high concentration of HCN in vapor form. The correlation of the *length* and *severity* of the exposure determines its adequacy to cause a given disease.

Obtaining the Occupational History.—The occupational history begins with the occupation in which the patient has most recently been engaged. The necessary details include name of occupation, length of employment therein, exact nature of work done (materials handled and mode of handling), hours of work in exposure, other hazards in the same room, environmental conditions, and protective devices if any. In many cases of occupational diseases the source of the illness will be found in the job *most recently* held. In others it will be necessary to inquire into previous jobs in order to locate the causative exposure. In any case it is advisable to get as much information as possible about previous employments and potential exposures to industrial hazards.

Evaluating Severity of Exposure.—When the occupational history has been obtained, the next step is to evaluate the severity of the exposure involved in the occupations named. If the physician has personally observed the given occupations and knows what materials are being used as well as the environmental conditions prevailing in the factory, he can make an accurate evaluation of the nature and severity of that exposure. Lacking this knowledge he must turn to other sources of information.

One source of aid is the *accumulated literature* on occupational diseases and on industrial processes. Another source is to be found in the *divisions* (or bureaus) *of industrial hygiene* in the Health Departments of twenty-nine states and several cities, and in the Labor Departments of two states. Where such state or local industrial hygiene units have not yet been organized, information can be obtained from the

Division of Industrial Hygiene, National Institute of Health, United States Public Health Service, Washington, D. C., which is the Federal center for research in occupational diseases.

Determining the Extent of Injury.—When the adequacy of the exposure has been established, the physician proceeds by appropriate *laboratory tests* to determine the extent of injury caused by the exposure. If silica is the hazard involved in a given case, for example, a roentgenogram of the patient's chest is indicated. When the history shows exposure to benzol, a complete hematologic examination is required, and where the history leads to the suspicion that an occupational source of irritation is the cause of a dermatitis, recourse may be had to patch tests.

The diagnosis of an occupational disease is therefore founded on a carefully taken history and it is confirmed by laboratory tests. Signs and symptoms furnish corroborative evidence of its accuracy.

Exclusion of Causes Outside of Employment.—One word of caution about the diagnosis of occupational diseases. Always remember to exclude causes outside of employment. Ivy poisoning has been contracted on hunting trips as well as in erecting telephone poles. Carbon monoxide has the same toxic properties whether generated in the furnace in the factory or in the gas range in the home. Therefore rule out the possibility of an extra-industrial cause.

Treatment.—The one principle in treatment which is applicable to all occupational diseases is *removal of the patient from the exposure responsible for his ailment.*. Otherwise, treatment is *symptomatic* being governed by the nature and extent of the pathology present. *Transfusions,* for example, may be indicated in the anemia of benzol poisoning, whereas the anemia of lead poisoning is seldom so grave as to demand that form of treatment. A knowledge of the excretion of poisons is sometimes a guide in treatment. Thus *purgation* is indicated in the treatment of lead poisoning because lead is excreted mainly by way of the bowels. Some poisons are cumulative. An attempt to obtain rapid excretion of a cumulative poison may mobilize dangerous amounts of it in the blood stream and bring on an acute poisoning. Most patients

will rid themselves of an accumulated poison satisfactorily if removed from exposure.

Prognosis.—*Silicosis* is the best example of an occupational disease in which it is possible to offer a reasonably accurate prognosis because a sufficient number of silicotic patients have been followed for a sufficiently long time to permit the acquisition of definite knowledge of sequelae. Similar information about other occupational diseases is rather scanty and indefinite. Industrial medicine still lacks long-term follow-up records of cases of occupational disease. The supplying of this lack would be a worthwhile contribution on the part of the general practitioner who alone has the prolonged contacts essential to accumulate such records.

Prevention.—Prevention of occupational diseases is a practical possibility in almost every case. The methods of prevention are well known. The advantages are obvious and are being recognized by employers, employees, and by the medical and engineering professions. The National Association of Manufacturers has organized a Committee on Healthful Working Conditions. The U. S. Department of Labor recently set up its Committee on the Conservation of Man Power in Industry. The American Medical Association has appointed a Council on Industrial Health. The National Safety Council is one organization through which the engineering profession participates in this common activity directed toward the reduction of the incidence of the ailments of occupation.

The physician's part in the prevention of occupational diseases begins with the *pre-employment physical examination* of all persons applying for work. The physician knows the requirements of the various jobs in the plant and is thus able to classify the applicants and see that each is placed in a job well within his physical and mental powers.

If the full value of medical protection for the plant personnel is to be realized, the health status of each employee must be *checked at regular intervals*. In this way the early signs of chronic disease are discovered and an opportunity is afforded to apply curative rather than palliative treatment

If the plant physician finds one or more cases of occupational disease among the employees in his plant, thereby

establishing the presence of an industrial hazard, he should immediately seek the services of an industrial engineer for the purpose of controlling that hazard. It is the engineer's duty to devise ways and means for such control. Engineer and physician together must then prevail upon plant management to install the necessary control measures.

After any apparatus for the control of an industrial hazard has been installed, the physician must check the efficiency of its operation by making frequent observations of the physical condition of those employees for whose protection the apparatus was provided. However efficient the protective device may appear to be, it still remains the duty of the medical man to determine its effectiveness, for his is the only profession whose members are qualified by training and experience to appraise the health status of the men and women employed in industry.

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INDUSTRIAL INJURIES

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This topic covers a wide range of subjects of constantly
increasing importance as the complexities and tempo of our
industrial life increase. Physiologic disturbances may result
from physical, chemical, thermal or electrical agents. Proper
handling of each type of injury is essential; first for the
preservation of life, and second for preservation of ultimate
function. This paper will deal with some of the major prob-
lems presented by industrial injuries.

FIRST AID

In any accident, medical and surgical aid should be ad-
ministered as quickly as possible consistent with the welfare
of the patient. Of prime importance is *immediate examina-
tion* to determine the severity of injuries and the order of
precedence of therapy.

Hemorrhage and Shock.—*Hemorrhage.*—Immediate
effort toward hemostasis is of primary importance. This is
best accomplished by direct pressure of a dressing over the
wound. Occasionally a tourniquet is advisable.

Shock.—The next consideration should be prevention of
shock or the care of a patient in shock. Injured individuals
should never be handled roughly, jerked about, or the injured
parts subjected to trauma. Immediate splinting is necessary.
The use of a board or stretcher goes far toward reducing
shock.

In *treating* shock in the ordinary individual, the patient's
head should be lowered and his feet elevated. In one with

a heavy abdomen, the fat and abdominal contents may place undue pressure on the diaphragm and embarrass an already feebly beating heart; such individuals do better in a horizontal position with only the head lowered. Maintain the patient's temperature by external heat and sufficient covering, for chilling will predispose greatly to a drop in blood pressure. Loss of fluids should be made up by transfusion and by intravenous glucose and saline solution; I prefer 5 per cent glucose in normal saline, or blood plasma may be used, but I believe the action of the patient's blood cells should first be checked against the plasma to be given. Administration of oxygen is of great help.[1, 2, 3] The drugs used fall into four groups: (1) analgesics or narcotics such as codeine, morphine and pantopon; (2) the peripheral vascular constrictors, such as adrenalin and ephedrine; (3) those drugs which affect cardiac function, such as the digitalis group; and (4) those agents affecting the respiratory center, such as carbon dioxide (which should *not* be used in a concentration of over 5 per cent lest it blanket oxygen in the alveoli).

Once shock is controlled, immediate surgery may be considered, as well as secondary care, and the use of chemotherapy to prevent infection. Such drugs as sulfanilamide, sulfathiazole and allied compounds may be employed in a prophylactic manner.

ANESTHESIA

General Anesthetics.—These have their place, but their use should be steadily diminished as skill in handling local, nerve block, or spinal anesthesia increases. The *intravenous* general anesthetics have value, but their toxicity—and corrective or resuscitating measures in case of untoward reactions—should be fully understood by those administering them. *Rectal* anesthetics have certain advantages, but they may be hard to control and usually need supplementing. In all general anesthetics, the hazard is in direct proportion to the *time element* or *length of anesthesia*. The inhalant varieties have some very distinct disadvantages in adding to shock, loss of fluid through sweating, lung irritation, nausea and vomiting, and too often a severe grade of anoxia occurs. I have seen the entire personality of an individual changed by

surgical procedures when severe, prolonged anoxia resulted from deep anesthesia, insufficient oxygen exchange and prolonged shock. General anesthesia too often prolongs convalescence.

Local Anesthesia.—Local anesthesia with *novocain* (procaine), often nerve block, is the method of choice.[4] *Spinal anesthesia* may be employed to great advantage in many surgical procedures, particularly in the lower back, abdomen, pelvis and lower extremities. Everyone employing this method should refer to the work of Maxson.[5]

Administration of a *barbiturate* with local or nerve block will do much to allay apprehension and tension suffered by some individuals in these procedures.

<center>HEAD INJURIES</center>

Too often these involve injury to the brain, and concerning such injuries Dandy[6, 7] counsels well against hasty and ill-considered procedures, emphasizes the necessity for thorough observation and study of each case and notes that *conservative* measures benefit the greatest number. He pointedly remarks "patients don't die from fractures of the skull, they die from injury to the brain; patients don't have epilepsy from injury to the skull, they have epilepsy from injury to the brain." He believes too much treatment is directed to the fracture and not the essential injury. He points out dangers of giving intravenous injections of hypertonic glucose or sodium chloride promiscuously; withdrawal of spinal fluid; rushing the patient back and forth for x-rays; and with regard to lumbar punctures, points out that brain injuries cause increased intracranial pressure, and that withdrawal of spinal fluid tends to increase the trauma proportionately. This I have observed in a number of cases.

Of particular danger is the release of tension through a spinal puncture when an *extradural hemorrhage* is present, as it then simply increases, further embarrassing brain cells and substance. Hypertonic solutions of salt or glucose introduced into the venous system may produce temporary relief, but the reaction is apt to be correspondingly severe.

Dandy's method is to let the patient strictly alone until

it is known that nature is unable to cope with the situation. Constant study is necessary.

Important Points in the Management of Head Injuries.—*State of Consciousness.*—This Dandy ranks as the most important observation. If unconsciousness deepens during the first five or six hours, this is a clear indication that intracranial pressure is increasing and that surgical intervention must be considered. If consciousness is returning, it is just as clear an indication that cerebral pressure is being reduced and the patient is improving.

Recording of Pulse, Respirations and Temperature.—The recording of these every ten or fifteen minutes and the charting of curves will clearly indicate whether increased intracranial pressure is being compensated. A slowing pulse and dropping respiratory rate indicate increasing intracranial pressure. An impending break in compensation is shown by variations in the rate of pulse and respirations, and the latter may become of the Cheyne-Stokes' type. A temperature below 101.5° or 102° F. is valuable indication of a patient's safety. Fluctuations in temperature may give the first clue to increased intracranial pressure. "When the temperature is steadily rising, you can be sure that that is the measure of intracranial pressure; blood pressure records are of little value."

Restlessness.—Restlessness, which is a clear indication of compensation for or a break in the toleration of intracranial pressure, may accordingly indicate relief or the onset of coma. It is the "twilight border" on either side. *Morphine should never be used* to allay restlessness lest it mask signs of increasing intracranial pressure.

Involuntary Micturition or Defecation.—Dandy states that incontinence of urine or feces when the patient is seemingly conscious means a break in cerebral compensation.

Treatment.—With regard to *operation*, less than 10 per cent of the patients require it, and 20 per cent may die regardless of therapy because of the severity of the injury. If a patient is going to die from injuries of the brain, *excepting* extradural hemorrhage, within less than five or six hours, little can be done to avert the outcome. Six or eight hours of observation are advisable, as a rule, and a break in com-

pensation must be followed (when warranted) by immediate surgery. Indecision and delay are then highly dangerous.

The first consideration in treatment is preservation of life; the second, preservation of function. These are guides in raising depressed areas of skull, treating fractures involving sinuses, subdural hemorrhages, subdural hydroma, air in the cranial chamber, and carotid-cavernous aneurysm.

Subdural Hematomas.—Immediate diagnosis and operative care are imperative. These cases may be recognized by the following criteria: The patient may walk about for fifteen to thirty minutes, then display symptoms of drowsiness followed by rapidly ensuing coma with decompensation in three to five hours, or he may "drag along," and develop paralysis following a convulsion beginning in the face, progressing to the arms and then to the legs; this sequence in an attack is always the same. Frequently paralysis develops in exactly the same order, namely, the face first, arms next, and finally the legs. Hemorrhage results from a crack in the bone which produces tears of the middle meningeal artery; once torn, as the hematoma increases, smaller branches are broken off which make the clot increase more rapidly in size. Or the symptoms may be mild, progress slowly for weeks or months, and end in decompensation and death. Thus writes Dandy.

Treatment is to expose and tie or thrombose the middle meningeal artery and evacuate the clot; this may be done through a small perforation on the left side if the patient is right-handed, over the temporal or parietal region. If no great quantity of blood is found the other side should be explored. If neither of these punctures provide relief from pressure, a right-sided subtemporal decompression is performed. If a clot is disclosed on either side, the decompression is made on that side.

Dandy adds a final note of warning on post-traumatic neuroses. He cautions against over-study and treatment of individuals in whom ventriculograms have revealed no source of intracranial pressure.

In cases in which fracture of the skull involves sinuses, early administration of sulfanilamide and sulfathiazole may be employed to great advantage.

INJURIES OF THE EYE

Angus L. MacLean, M.D.[1]

Burns.—Thermal, caustic and chemical injuries can cause much pain, lacrimation and chemosis, with swelling of the eyelids. The injury may be transient and superficial, or may cause dense deep opacification; and perhaps symblepharon or perforation due to sloughing, and even shrinkage of the eyeball. Damage is more intensive and repair later after alkali than after acid burns. In order of severity are burns from caustic soda, hydrochloric, sulfuric, nitric and acetic acids. Ammonia may cause very serious injury. Superficial glancing injuries from thermal agents are less serious than chemical injuries. After superficial ones, there is hypersensitiveness; with severe burns insensitiveness and necrosis are likely.

In *caustic burns* (alkali), promptly irrigate with slightly acidulated boracic water, 1 per cent tannin or 1 per cent acetic acid to neutralize the caustic soda, and instill ½ per cent pontocaine to relieve pain; boric irrigations should be repeated. If the burn scars the cornea, the patient should be admitted for immediate paracentesis. Acetic acid, 1 per cent, should be instilled hourly for the next twenty-four hours; the precipitated proteins are not soluble, so do not penetrate the cornea farther than the initial burn; it is therefore best not to neutralize them. Instill ½ per cent pontocaine and irrigate again with boracic acid solution.

Lime burns may cause a deceptive necrosis and may leave dense opacities. In early cases instill cocaine, holocain or pontocaine, thoroughly remove and wash out the free lime and irrigate copiously and long with 2 per cent chloride of ammonium or 10 per cent neutral ammonium tartrate, after which insert petrolatum album; the eyelids may be lifted with light retractors.

Following emergency measures, all cases should be treated in the usual manner for traumatic conjunctivitis or corneal ulcers. A bland soothing ointment, such as 2 per cent butyn, with metaphen 1:3000, should be applied frequently to relieve pain; if necessary these should be augmented by instillations of pontocaine, butyn or holocain. An eye pad should be

maintained and changed frequently and the boric irrigations repeated until the corneal abrasion has healed.

Physical Agents.—*Injury by Cold, Wind and Light.*— Epithelial edema and exfoliation may rarely arise from exposure to cold. After preliminary lacrimation, absence of blinking tends to drying and wind contributes to the injury. Recovery occurs in three to ten days. Instill pontocaine and apply a bland ointment dressing.

Excessive exposure to ultraviolet light, as in bright snow (producing "snow blindness"), in cinema studios, in electric welding and medically in mercury-vapor lamp treatments, causes photophthalmia—a combined reaction of the eyelids, conjunctiva and cornea (actinic keratitis). After a latent period, acute distress occurs, featured by pain at times "neuralgic," photophobia, lacrimation, blepharospasm, hyperemia of the interpalpebral bulbar conjunctiva, edema and in extreme cases erosion of the corneal epithelium.

Foreign Bodies.—A cinder under the *upper lid* usually lodges in the slight sulcus just above the lid margin and causes pain by being constantly rubbed over the cornea by winking movements of the lid. To remove it, evert the lid with gentle traction of the lashes and downward pressure with a small wooden applicator on the upper part of the lid. Remove foreign substances with a small cotton applicator. The lid is then returned to its normal position by having the patient look upward. Local anesthesia is not necessary unless the eye is very sensitive.

A foreign body on the *cornea* causes pain each time the lid passes over it. If not removed for some time, it may become embedded and lead to ulceration. To remove, instill a few drops of ½ per cent pontocaine solution and repeat in one minute. The foreign body can be removed with a sterile toothpick or a metal corneal spud. All remaining débris should be carefully scraped and cleaned away. A particle of steel or iron should be removed with a small hand or pencil magnet. For deeply embedded cases, apply a bland ointment and pad for twenty-four hours.

Intra-ocular foreign body is a major injury and may cause complications with permanent impairment of vision. An x-ray examination for diagnosis and localization should al-

ways be made. A particle of iron or steel can be removed readily by magnet extraction. If it is lodged in the anterior chamber the approach should be through a small incision in the cornea; if in the vitreous chamber, through a small scleral incision opposite the point of x-ray localization. If nonmagnetic and in the vitreous chamber, extraction is usually impossible. It may become encapsulated and remain dormant for years but usually produces a severe purulent destructive inflammation requiring enucleation.

Lacerations.—*Small* ones of the *lids* and *eyeball* should be repaired under local anesthesia, novocain infiltration for the lids and pontocaine instillations for the eyeball. The skin is cleansed in the usual manner with soap and water, followed by alcohol. Care must be taken to prevent contact of alcohol or soap with the eyeball.

Through-and-through lacerations of the lid should be closed in two layers. First, the tarsal conjunctival gap is closed with interrupted buried No. 000000 catgut sutures, these passing into the tarsus but not through the conjunctiva to the posterior surface of the lid. The skin is then closed with interrupted fine silk sutures. To prevent notching at the lid margin, the conjunctival and skin suture lines should not overlie each other—this result is accomplished by Wheeler's "halving" technic.

Lacerations of the *cornea* and *sclera* should be closed with interrupted black silk sutures, using a very fine special corneal needle and extra fine thread, passed through the superficial half of the cornea or sclera close to the edge of the wound. Corneal lacerations are usually accompanied by prolapse and incarceration of the iris. If it is torn or cannot be replaced, the prolapsed portion must be excised and cut edges replaced in the anterior chamber. If the laceration is extensive, and involves the anterior chamber angle and ciliary body, early enucleation must always be kept in mind owing to the danger of sympathetic ophthalmia.

Superficial corneal abrasions are detected by instilling a drop of 2 per cent fluorescein on the cornea and irrigating it with normal salt or boracic acid solution. The abraded area will appear green, while the normal cornea remains clear and unstained. Treat with a soothing bland ointment and pad

until healed. As a rule, unless Bowman's membrane has been traumatized, no permanent opacity or scarring remains.

INJURIES OF THE CHEST

The rib cage, with the sternum in front, the spinal column in the back and the diaphragm below, surrounds complicated mechanisms and trauma may produce: (1) hemorrhage or hemothorax; (2) pneumothorax; (3) a combination of the two, with consequent embarrassment of respiration and cardiac action; (4) injury of the diaphragm, with herniation of abdominal viscera; (5) injury of the heart or pericardium; (6) infection of the pleural cavity through the injury or by the bronchial route.

Classification.—Connors[9] classifies these acute injuries of the thorax as either *open* or *closed* wounds. The former involve only the chest wall and are extrathoracic, while the latter involve the pleura, lungs and possibly the diaphragm and constitute an intrathoracic injury. These wounds may be classed as *nonpenetrating* when the skin, muscles and subcutaneous structures are involved without opening the pleura; *penetrating* when it is opened.

Contusion may be associated with extensive hematomas, with rib fracture, and with pleurisy and effusion. Connors states that 10 per cent of all visceral chest wounds are this type of injury. The symptoms are pain on inspiration, friction rub and slight fever. Progress is generally good after strapping of the chest with adhesive.

Concussion of the chest may result in slight or gross disturbances, depending on the degree and type of trauma. It is usually associated with sharp blows, resulting in profound reflex stimulation of the sympathetic nerves, and is similar to cerebral concussion. Symptoms vary from vertigo to all the symptoms and signs of shock; death may occur. Treatment should be symptomatic and supportive.

Contusions of the lungs may occur with or without rib fracture. Diagnosis is made from the presence of hemoptysis, fever, and signs of consolidation in the lungs, and from the x-ray findings.

Subcutaneous laceration of the lung is caused by the end of a broken rib. Hemothorax and pneumothorax may both

be present, and shock may accompany this injury. Diagnosis can be made on clinical findings and by x-ray. Connors advises conservative treatment and cautions close observation of the patient for development of tension pneumothorax and occasionally for acute anemia; he advises intrapleural pressure readings at frequent intervals.

Traumatic subcutaneous rupture of the lungs is infrequent. Again one may find no cutaneous marks, and fractures of the ribs may not be present. The pleura may be damaged, allowing blood and air to escape into the chest cavity and resulting in hemopneumothorax, with the usual signs. Mediastinal emphysema develops if the mediastinum is torn. Infection is very prone to occur in these cases. Connors advises puncture of the chest wall and operation to (*a*) close the pleural cavity and stabilize the mediastinum, (*b*) control hemorrhage and (*c*) drain the infected area. He suggests exteriorization of the lung by suturing it to the chest wall and packing the laceration. Relief of tension pneumothorax and mediastinal emphysema may demand urgent, instant attention. Mortality with or without operation is very high. The prognosis is better with a certain diagnosis and operation.

· *Compression of the chest* lasting for a few minutes in severe crushing injuries produces cyanosis, apnea and pressure stasis. Traumatic asphyxia results. Respirations may be short, superficial and irregular with a weak rapid pulse. There is general pallor with areas of purple discoloration, the skin is cold and clammy, and the patient may become stuporous. Treatment is given as for shock, with oxygen, intravenous injection of normal saline solution or transfusion, and general care. The prognosis is good if patient survives the first hours of injury.

A second type, seldom encountered, is less serious but may be fatal. This type is characterized by diffuse ecchymosis of the face, neck and upper thorax due to a sharp sudden rise of intrapleural and mediastinal pressure, forcing blood back into the valveless veins of these regions. Petechial hemorrhages, exophthalmos, eyelid edema and conjunctival hemorrhages may occur, with the color later changing to a vivid

purple, and a sharp line of demarcation at the border of un-involved arms, chest and body. With continued pressure, submucous and subcutaneous hemorrhages may occur, as well as visual disturbances due to optic atrophy. Connors suggests rest in bed and oxygen inhalation. Recovery takes place spon-taneously within seven to eight days if there are no complica-tions. I have seen two of these cases; the appearance of the patients leaves an everlasting impression.

Traumatic emphysema spreads from the wound area, and may involve the entire body including legs and genitalia. The skin may rupture from distention. Crepitation is elicited and air is demonstrable by *x*-rays. The wound should be treated and skin tension relieved by incision or large trochar.

Active mediastinal emphysema may result quickly in death from pressure on the great vessels unless immediate relief is given. Emphysema appears above the sternum and the clavicles, and ranges upward and outward; marked cardio-respiratory disturbance occurs. Immediate operation through a suprasternal incision is advised. A large drainage tube is inserted down to the trachea, converting a closed pneumo-thorax to an open one. Intrapleural pressure readings are made, with relief of tension pneumothorax by aspiration, and in urgent cases intercostal incision may be necessary.

Traumatic pneumothorax may be unilateral or bilateral, with partial or complete collapse of the lungs. Air may enter from a bronchus (closed pneumothorax), or through the chest wall (open pneumothorax). If the condition is unilateral, the mediastinum is pushed over to the opposite side, reducing the vital capacity of the remaining lung and causing respira-tory and circulatory difficulties.

Tension pneumothorax occurs in traumatic subcutaneous rupture of the lungs with compression of the chest while the glottis is closed. Shock, cyanosis, dyspnea and early medi-astinal emphysema occur. Coughing increases distress. Thoracentesis with a needle releasing the air subaqueously is advised, with repeated estimations of intrapleural pressure.

Large external open pneumothorax may cause mediastinal flutter and, if both sides of the chest are open more widely than the tracheal openings, collapse of both lungs occurs with

apnea and death unless the lungs are immediately inflated. Flutter may be prevented during operations by steadying the lung with a lung forceps. Connors advises closure and fixation of the lung if necessary.

Traumatic hemothorax may be unilateral or bilateral, and may be present in closed thoracic wounds; it is often combined with pneumothorax. In severe cases blood may occur in the contralateral side. The lung is partially collapsed, or shrinks to small volume. The diaphragm is high and immobile but a large hemorrhage may push it flat. The mediastinum is displaced to the uninjured side. The blood does not coagulate and I have seen the surface ripple in fluoroscopic examination. It absorbs slowly, and may act as an irritant causing pleurisy with exudation. Pain, cough, dyspnea, possibly hemoptysis, fever and weakness occur. The physical signs are those of fluid in the chest. *x*-Ray plates or fluoroscopic examination and thoracentesis confirm the diagnosis. Infection is the most serious complication. Rest in bed and sedation are usually sufficient. In moderate hemothorax early aspiration with replacement of air is advised, or aspiration without oxygen replacement after seven to ten days. Aspiration must be high since the diaphragm is often elevated. I have seen one striking case of hemopneumothorax following the lifting of a heavy ledger in which the symptoms at first simulated those of fractured rib, but later signs of shock appeared, with fluid in the chest, then evidence of hemorrhage from which patient died after exhibiting typical signs and symptoms of cerebral anemia. At autopsy it was found that a small subclavicular adhesion had been torn through, rupturing a small artery which continued to spurt until the right side of the chest was almost completely filled with blood.

Massive collapse of the lungs may follow chest injuries, and can involve one or both lobes or a whole lung. Connors states that carnification occurs in one type in which crushing injury to the parietes occurs with intrabronchial mucus without direct injury to the affected lung, and he advises excision lest anaerobic infection and gas gangrene of the lung result.

Traumatic pneumonia is rare.

Traumatic hernia of the lung also is rare. Strangulation may occur. The treatment is operation.

ABDOMINAL TRAUMA

SAMUEL McLANAHAN, M.D.[20]

In abdominal wounds indirect violence may play an important role. With neither bruise nor scratch of the skin on the abdomen, an internal injury of serious degree may be present, with signs of such an injury delayed for hours or days. Late hemorrhage from a ruptured spleen is illustrative. Diagnosis must be guarded in an accident where severe trauma *may* have resulted.

Abdominal wall injuries are generally obvious, but a puncture wound may cause a rent in the peritoneum as well, and demands an early and unequivocal answer. Hematoma of the rectus abdominis muscle is likely to be confused with a lesion which is intraperitoneal; though potentially not as serious, it may be quite as painful. Evacuation of the trapped blood and blood clot is frequently advisable.

Penetrating or nonpenetrating wounds may damage the abdominal contents, and result in *hemorrhage, perforation* and *shock.* Puncture may occur through the perineal route as well as the more likely anterior abdominal wall area. Treatment of shock with prompt hospitalization and early surgical exploration is in order as soon as the patient's condition permits. The *nonpenetrating* or "subcutaneous" injuries are difficult. A ruptured viscus will at once release gas which may give an early lead toward diagnosis, and also obliterate the area of liver dulness on percussion. Air, usually beneath the diaphragm, may be seen in a flat roentgenogram. Release of gastro-intestinal contents usually produces immediate peritoneal irritation.

Intra-abdominal hemorrhage is most likely from tear or rupture of the liver or spleen, but may arise from a mesenteric tear, which may be hard to diagnose. A small hemorrhage may cause great shock, therefore few clinical differentiations are more difficult than that between hemorrhage and shock in abdominal trauma. Experienced clinical judgment and careful and repeated observations are important. Signs of increasing peritoneal irritation coupled with a falling red cell and rising white cell count are significant. Fluids administered intravenously generally improve shock but may fail to do so in hemorrhage. If after a period of observation re-

sponse is not satisfactory, and the patient's condition permitting, exploration must be done. Whole blood and blood plasma are essential aids in such an emergency.

In such exploration the operator must be prepared to remove the spleen, suture the liver or resect the intestine. Suture of the spleen is only rarely practicable and its complete removal is usually required. If suture fails to control hemorrhage liver packing must be resorted to, though such a procedure carries new dangers with it.

Intestinal injury *from compressed air* deserves a reference. A workman in playful mood applies a compressed air hose nozzle to, or near the anus of a fellow worker, with resulting sudden forcible dilatation of the colon. Laceration may occur, followed by fatal peritonitis. This injury has been reported repeatedly with high mortality. Workmen using compressed air should be warned against the dangers of such a "joke."

Traumatic appendicitis is a well recognized entity but only under conditions such as freedom from previous complaint, direct severe trauma to the region involved, indirect trauma of unusual severity, and onset of characteristic symptoms very soon after injury. As one writer notes, "Pain, Prompt, Persistent, Progressive (the four P's)." With these, treatment is appendectomy.

INJURIES OF THE GENITO-URINARY TRACT

HOWARD C. SMITH, M.D.[1]

Injuries of the Kidneys.—Although the kidney is fairly well protected by its position, mobility and surrounding fat pad, injuries may occur and are due to direct or indirect trauma. Indirect trauma from blows to the side or back, falls from heights onto the feet or buttocks and violent muscular efforts, as well as penetrating wounds, must be considered. Spontaneous rupture of the kidney is rare and is usually associated with some pathologic condition.

SYMPTOMS AND DIAGNOSIS.—Most important is *hematuria*, but this may be absent due to severance of a ureter, blockage by blood clot, or reflex anuria. *Shock, pain in the flank*, a *mass* and *rigidity* are usually present. All symptoms

vary with the severity and type of injury. Blood pressure and pulse rate must be carefully watched, the pulse being taken every fifteen minutes and blood pressure every half hour until the patient is out of danger. Some degree of shock is almost always present in severe injuries.

The history of injury is important. If the patient is unable to void, catheterization under the most careful aseptic technic should be carried out, followed by intravenous urography when the patient's condition permits. Cystoscopy is more hazardous owing to danger of infection, but may be indicated.

TREATMENT.—Treatment is palliative in the large majority of cases. Patients should be grouped and matched for transfusion as soon as possible. If marked increase in pulse rate or fall in blood pressure is noted, operative interference should not be delayed. The type of operation depends on the findings and judgment of the surgeon. If nephrectomy is indicated, one must be sure the opposite kidney is present. Its presence may be ascertained by opening the peritoneum and palpating it before the injured kidney is removed.

Injuries of the Ureters.—Because of its small size, position and mobility the ureter is rarely subject to traumatic injury. The majority of injuries are due to operations and intra-ureteral instrumentation.

SYMPTOMS AND DIAGNOSIS.—Urine extravasated into the adjacent tissues causes *pain*. Accidental unilateral ligation may go undiagnosed unless infection is present, urinary extravasation occurs or a fistula develops. If injury occurs during the making of a ureteropyelogram there is acute pain after 2 or 3 cc. of solution have been injected, and the procedure should be stopped. An x-ray plate will clinch the diagnosis. *Nausea* and *vomiting* are common but may subside after twenty-four hours. *Tenderness, rigidity* and *tumefaction* develop shortly, as well as *abdominal distention.*

Intravenous urography is most important in diagnosis. Cystoscopy may be indicated but it is dangerous owing to risk of infection. If the ureter can not be catheterized, intravenous injection of indigo carmine is helpful.

TREATMENT.—If the injury is due to a surgical procedure, immediate repair is ideal. This may require a uretero-

ureteral anastomosis over a catheter, a ureteroneocystostomy, or ligation of the ureter if the injured kidney is uninfected and the other kidney is normal. In case of rupture by intra-ureteral instrumentation, if the urine is uninfected, splinting with a ureteral catheter for five or six days is sufficient. If urinary extravasation and sepsis result, immediate surgery and proper drainage are indicated.

Injuries of the Bladder.—These may be divided into three groups: (1) *contusions,* clinically important in differentiation from rupture; (2) *wounds,* which are produced by perforation and usually communicate with the exterior; (3) *rupture,* the most common and a real emergency.

SYMPTOMS AND DIAGNOSIS.—Crushing injuries involving fracture of the pelvis may be accompanied by *rupture* of the bladder. Symptoms of intraperitoneal or extraperitoneal rupture are similar but are more severe in the former type, with a *marked desire to void* without being able to pass more than a few drops of blood. *Hypogastric pain* and signs of *peritonitis* are present in intraperitoneal rupture.

Intravenous urography is of great help in establishing a correct diagnosis. Catheterization to confirm an empty bladder or the presence of small amount of bloody urine is helpful diagnostically but introduces risk of infection. Instillation of a measured amount of sterile solution into the bladder through a catheter and measurement of the recovered amount is of help. Air cystography has been advocated, but carries some risk of air embolism.

TREATMENT.—Early diagnosis and prompt surgery are essential except in contusions. Supportive measures, such as transfusions, heat applications and stimulants, are indicated. Immediate suprapubic exploration should be done if there is doubt of the diagnosis. If no extravasation is found extraperitoneally, the peritoneum is opened and the peritoneal surface of the bladder examined. If no infection is present, the tear is repaired and the peritoneum closed without drainage. The bladder should always be drained with a double suction tube whether the rupture is intraperitoneal or extraperitoneal, and should be left in until healing has occurred. Injuries involving the floor of the bladder with resulting deep pelvic extravasation may require perineal as well as supra-

pubic drainage. Important in urinary extravasation is prompt and adequate drainage. In case of infection the sulfonamide group of drugs should be used, the proper one being indicated by the organism cultured.

Injuries of the Urethra.—Injuries of the male urethra consist of (1) *external* wounds, (2) *intra-urethral* injuries and (3) *rupture* of the urethra. The first type, which is uncommon, may be produced by a cut, stab or splinters. The second is due to instrumental trauma, foreign bodies or burns by chemical agents. The third is caused by "straddle injuries," or by blows or kicks in the perineum; it also occurs with fracture of the pelvis.

Symptoms and Diagnosis.—Early, accurate diagnosis is essential and prompt treatment is necessary if complications and infection are to be avoided. Most important are the history and a careful physical examination. *Pain* in the perineum, *retention, bleeding* from the meatus, perineal *hematoma* and *extravasation* are the chief symptoms and signs. Rupture of the posterior urethra is accompanied by profound shock, and death occurs in 40 per cent of cases.

Treatment.—Treatment of *rupture of the anterior urethra* varies. If extravasation has not occurred and the injury is not too extensive, splinting with a urethral catheter may suffice. A retention catheter predisposes to infection and stricture formation. In all serious injuries, diversion of the urinary stream and adequate drainage of the extravasated regions are of the utmost importance. With severe shock, supportive measures must be instituted and operation postponed.

In *rupture of the posterior urethra* a catheter should be introduced into the bladder, for strictures are not as prone to occur in this area. Occasionally this may be accomplished by merely passing it through the urethra. Other cases require perineal operation or combined perineal and suprapubic cystostomy in order to attain this objective. Davis (1934) brought out a set of sounds, male and female, which are of great help in getting a catheter through the ruptured area after suprapubic cystostomy. The catheter should be left in place until the rupture has healed, which usually means two or three weeks.

All cases of ruptured urethra should be followed for at least eighteen months to make sure that no strictures develop. Patients who are operated on many months after rupture of the urethra, or who have it torn off at the apex of the prostate, are difficult cases for a urological surgeon. Early operation is the ideal treatment.

INJURIES OF THE SKELETON AND EXTREMITIES

In this machine age the surgeon is confronted with a variety of injuries to the organs of locomotion, which for variety, bizarre character and extent often simulate war wounds. With our present knowledge of surgical technic, with trained surgeons available throughout the country, excellent antiseptics, chemotherapy, rapid transportation and hospital facilities, the injured individual deserves more than half-way measures and compromise therapy.

Chemotherapy.—In July of 1937, I[12] published the first cases of gas gangrene treated with *sulfanilamide*. Following this, prophylactic and therapeutic use of this drug developed rapidly in the treatment of traumatic wounds, and has been of great value in saving limb and life. The powdered form[13,14] may be dusted into the wounds after their thorough toilet and lavage. I have used it in this manner in about 100 cases. It produces a severe chemical reaction, hence it is very necessary to watch circulation after this procedure. It raises the local concentration to a high percentage, stopping all local bacterial action. It definitely delays healing of both soft tissues and bony structures, is slowly absorbed from the area, and may give a concentration of 5 mg. per cent or more in the blood. The individual should be observed for toxic effects and if necessary the wound re-opened and lavaged. This danger is less likely and there is less difficulty with swelling, if suitable wounds are left open and packed with vaseline gauze after the method of Winnett Orr.[15, 16, 17, 18]

Sulfanilamide may also be given by mouth; or, if the patient is unconscious, unable to swallow, or nauseated, it may be given parenterally or per rectum. The concentration in the blood should be raised to an effective level, 10 mg. per cent, when there is danger of extensive or severe infection.

The *toxic effects* of the sulfonamide drugs should be thoroughly understood and these effects balanced against therapeutic values. Daily blood counts should be made, also daily determinations of sulfanilamide blood concentration. The latter can be increased by limiting fluids in adults to 1800 cc. each twenty-four hours, and likewise the concentration relieved by forcing fluids, use of intravenous glucose and normal saline, or ⅙ molar sodium lactate solution. If a moderate hemolytic reaction is noted, transfusion should be resorted to after careful grouping and matching, and repeated as often as necessary. If these safeguards are followed, few individuals show a severe toxicity contraindicating the use of sulfanilamide.

The same principles apply to *sulfathiazole* and the various other sulfonamide drugs. The work of Long and Bliss,[10] as well as that of Mellon and co-workers,[20] should be consulted for the use of these compounds; or a shorter article is informative.[21] Sulfanilamide and its allied compounds have been well proved of great value in the ordinary wound infections and in gas gangrene, both from the prophylactic and the therapeutic viewpoints, but in all cases this therapy must be combined with sensible surgical procedures, observing the principles which govern circulation, drainage, restoration of position, fixation or immobilization, repair or healing, and problems associated with general care and nursing of the patient.

Open Wound Therapy.—Winnett Orr[15, 16, 17, 18] has for years pleaded and demonstrated these principles. His work has been given the acid test of war by Trueta.[22, 23, 24]

Compound fractures, if there is more than a puncture or small opening, should be thoroughly washed out with normal salt solution and if greasy foreign material is present, with ether, avoiding sclerosing solutions. All foreign matter should be removed, but all obviously damaged areas need not be cut away; I believe far too much débridement is done. A surprising amount of tissue will survive if circulation is preserved and the various spaces of the wound are thoroughly packed with vaseline gauze, which is the next step in the procedure. If the fracture be adequately reduced and immobilized with plaster properly applied, much pain and dis-

comfort will be avoided for the patient, his hospital stay will be materially shortened, and his recovery hastened. Tension must, however, be avoided. Should adherent or unsightly scars remain, a plastic operation is in order later.

Wounds of *soft tissues* with ragged lacerations, contusions and macerations from trauma may be treated in like manner. The vaseline gauze packs are extruded as the wounds heal. This therapy has the disadvantage of being rather smelly but when properly performed yields amazing results. All affected structures should be inspected and those sufficiently damaged should be repaired. It is especially necessary to search for damaged tendons and nerve trunks. Adequate dosage of tetanus antitoxin should be administered early; gas gangrene antitoxin, I believe, is of little value from a prophylactic standpoint. Some wounds may be closed tightly, but others require drains; sterile rubber bands answer this purpose well. Wounds associated with compound fractures should not be drained.

Closure of Wounds.—Clean *lacerations* may, if thoroughly washed out, be sutured, provided circulation is not endangered by tension or a number of great vessels have not been destroyed.

The closure of *compound fractures* is dangerous. Lenhard,[25] head of an active fracture service, has not closed a single compound fracture in thirteen years and in this time he has not seen a case of gas gangrene. I have repeatedly seen gas infection resulting from impaired circulation. Tension with obliteration of capillary circulation is the greatest single predisposing factor. Circulation may be obliterated by swelling from the injury, in which case relief incisions (always longitudinal in the extremities) should be made, the incisions being carried through the skin and fascial planes for complete relief of all tension. To avoid tension, fractures should be reduced, tight dressings avoided and, at the time of wound toilet, a thorough check of the basic circulation of the area made. Where the extremity has been too badly destroyed, amputation cannot be avoided. Once extensive thrombosis occurs, amputation is imperative. Thrombosis may result rapidly from improperly applied dressings, tension and internal or external pressure.

Gas Infections.—Sulfanilamide is specific for gas infections. In a recent compound comminuted fracture with merely a puncture wound, gas infection resulted from an improperly applied plaster splint, although the patient had received sulfanilamide from the onset of his injury but in a low dosage. Bacillus welchii heavily infected the wound, and much crepitation was noted from gas in the tissues of the affected area in the lower leg. All pressure dressings were removed; a massive dose of sulfanilamide was given, 80 gr. followed by 20 gr. every four hours, with an equal amount of bicarbonate of soda, rapidly raising the patient's blood level to 10 mg. per cent. The injury had been so extensive inside his leg that fascial planes were torn and decompression was automatic. The posterior half of the leg was held in a plaster trough and the patient was watched carefully for spread of gas. The entire area subsided in the space of a few days following the increased sulfanilamide dosage; surgical interference was unnecessary. The leg has since healed with good union of bone and the patient is walking.

In most cases, it is necessary to make incisions for the relief of tension; these may be packed with vaseline gauze and the extremity fixed with a plaster trough or skeletal support of some sort. Gas infections are more or less momentary trouble makers and tend to subside in four or five days under adequate therapy, or as other bacteria invade the wound. Fortunately, with chemotherapy, these secondary invaders are held in abeyance. Should plastic operation be done later on areas which have been infected by gas bacilli, one may find the spores lingering in the scar tissue and a fresh infection may light up unless sulfanilamide is used. It may be employed locally or systemically, or by both methods combined.

Fractures.—For reference work one suggests that of Böhler,[26] Watson-Jones,[27] and Key and Conwell.[28] (For infections of the hand, Kanavel.[29])

Joint Injuries.—Joint injuries may be classified as *penetrating* and *nonpenetrating*. In the latter, aspiration and air injection are of value.[30] Danger of air embolism is slight with proper technic. Injected air cushions the synovial membrane, is compressible, makes for comfort, and reduces the tendency to recurrence of hemorrhage or effusion. In *penc-*

THE CARE OF THE UNCONSCIOUS INJURED PERSON

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THE aim of this paper is to outline the procedures neces-
sary in caring for patients unconscious because of injuries to
the head. The unconsciousness may be caused by one or a
combination of several lesions, as classified later. An attempt
to determine the type of lesion must first be made. Starting
with the fact that the patient has been in a violent accident,
it must first be determined to what extent the signs and symp-
toms are due to injuries of the head or to injuries elsewhere.
The general condition of the patient as to injuries other than
of the head and the recognition of shock must be noted. With
evidence of shock there may be other signs simulating those of
severe concussion, leading to the conclusion that the major
injury is in the head when, as a matter of fact, the shock
may be due to a severe chest injury or concealed abdominal
bleeding. The frequency of an associated cervical spine
injury has led to the advice by Coleman[1] that x-ray examina-
tion of the cervical spine be included with that of the head.

DETERMINING THE SERIOUSNESS OF THE INJURY

State of Consciousness.—In considering the state of
consciousness, it is not sufficient to say that the patient is
unconscious. One must go further and attempt to determine
the type of unconsciousness. Delirium and coma represent
the main types.

Delirium is probably due to an irritative lesion. Such
lesions may range from slight cerebral circulatory disturb-
ances, as in concussion, to edema or extravasations of blood

into the brain or subarachnoid space in a quantity insufficient to produce increased intracranial pressure. It is important to recognize these irritative, low intracranial pressure types. In some the pressure may be almost at zero.

Coma signifies a state of unconsciousness in which the patient does not react to any type of stimulation. In a large majority of cases it is due to an increased intracranial pressure and there will be associated with it other signs of pressure and stertorous breathing. There is another type of unconsciousness in which the patient cannot be aroused though he presents none of the signs of increased intracranial pressure, and by manometric measurement there is no increase in the cerebrospinal fluid pressure. I refer to the *lethargic state* seen in the acute stage of traumatic encephalitis. This lethargy can further be differentiated from the coma due to increased pressure by its duration, for it commonly lasts over a period of three or four weeks, and in one of our recent cases continued for twelve weeks. Depending upon the location of the extravasations in traumatic encephalitis, the patients may be in an irritative or a stuporous state and these states may alternate from time to time.

Respiration.—In addition to the state of consciousness, the most important early indication of the seriousness of the lesion is the type and rate of respiration. It must be remembered that patients may remain unconscious for weeks, but a labored, markedly accelerated, or a very slow respiratory rate can exist for only a limited period. The respiratory difficulty may be central or peripheral in origin. A gradually increasing intracranial pressure, for example, may depress the respiratory rate in its early stage to 8 or 10 per minute. In the terminal stage of unrelieved intracranial pressure, the rate will be very rapid. In the delirious patient without increased intracranial pressure, the respiratory rate will be accelerated, often to a very high level.

Temperature.—The temperature range even in the early stage is a most important guide in diagnosis. An early sharp rise within an hour of the accident indicates disturbance of the thermoregulatory centers which may result in a state of hyperpyrexia, with a very rapid pulse and respiratory rate, constituting one of the most serious types of head injury.

The temperature range is most important in patients with bloody cerebrospinal fluid, indicating a sterile irritative meningitis. Here the temperature is usually under 102.5° F. rectally. This elevation is observed about forty-eight hours after the accident when there has been time for reaction on the part of the membranes. A high temperature not extending into the level of hyperpyrexia may be due to infection, but more commonly it is due to small hemorrhages in or about the brain stem.

Pulse and Blood Pressure.—The pulse rate will be low in cases with increased intracranial pressure, such as is seen in patients with large clots at any site within the cranium. In the terminal stage of increased intracranial pressure, however, the pulse, with the respirations, will be elevated, and the temperature may reach a high level. A slow pulse rate without other signs of increased intracranial pressure may occur in simple concussion. The rate in these cases may be as slow as 40, and this state may continue over a period of many days, but it is of no moment if the other signs of increased intracranial pressure are absent. On the contrary, a rate in the low 60's or high 50's, if associated with other signs of increased intracranial pressure, may demand the immediate evacuation of a clot. The pulse rate is accelerated in the meningeal irritative cases. Marked acceleration (140 to 180) may occur in cerebral irritative cases due to a disturbance of the cardiac regulatory mechanism.

Severe alteration in the blood pressure, persisting for more than a few hours after the accident, is a very grave sign. Slight variations in blood pressure, however, are unimportant and bear no relation to the level of intracranial pressure.

Neurologic Status.—The state of consciousness has already been stressed as one of the most important factors in determining the degree of injury to the nervous system. This is so because of the extreme value afforded by the knowledge of whether the conscious level is changing. Frequent examination will enable the observer to evaluate the patient's reaction to external stimuli. A diminishing response is a bad prognostic sign and usually indicates increasing intracranial pressure. When a patient first comes under observation, it is important to check the size of the pupils and their reaction to

light. A dilated pupil may indicate a clot or brain injury on the same side, and bilaterally dilated fixed pupils almost always indicate a fatal outcome. The extraocular movements cannot be tested fully, but the position of the eyes will often indicate an extraocular palsy. Conjugate deviation of the eyes may suggest an irritative lesion in the ipsilateral cortex.

The facial movements can be determined by pressure on the supraorbital ridge. The pharyngeal reflex will often indicate the degree of stupor. The motor status cannot always be determined accurately, but it is important to know to what extent the extremities can be used. The decreasing motor activity of one side of the body often indicates active intracranial bleeding. The deep tendon reflexes vary a great deal, from complete loss in profound coma to hyperactivity in the irritative states. The important factor pertaining to reflexes is inequality on the two sides of the body.

The presence of pathologic reflexes, such as the Babinski sign, may further indicate the site of a focal lesion. Jacksonian or generalized convulsions are not infrequently seen, and when present are indicative of cortical irritation. Bilateral tonic or clonic extensor spasm may indicate widespread injury. Occasionally extensor rigidity may be maintained over a long period and any external stimuli will provoke a convulsive seizure. Cervical rigidity and Kernig sign frequently suggest the presence of meningeal irritation from blood.

Skull Injuries.—*Linear fractures* ordinarily are of no importance, but they may extend into infected regions, such as the accessory nasal sinuses, or they may pass into the neighborhood of important vascular structures injuring, for example, the meningeal artery or the dural sinuses, resulting in the formation of extradural clots. Linear fractures may also extend into bony foramina in which cranial nerves lie, producing a tear of a nerve or a hemorrhage into its substance. The nerves most commonly involved are the optic, the facial and the auditory. There is often a loss of smell, extending over a long period of time. Unilateral blindness occasionally follows fractures of the frontal bone, extending to the apex of the orbit. The blindness is immediate and complete, and atrophy of the nerve ensues. Injuries to the

seventh and eighth nerves may be partial or complete. They tend to clear up over a long period of time, but may be permanent.

A most important linear fracture is that which extends through the base of the skull and is associated with a rent in the dura, resulting in cerebrospinal fluid *rhinorrhea* or *otorrhea*. The leakage of fluid through the nose may be entirely overlooked if the patient is in the recumbent position. The fluid escapes into the pharynx and in unconscious patients may cause very serious respiratory difficulty, but in patients in whom there is sufficient reflex it will be swallowed. To diagnose the condition, therefore, the patient should be placed in a sitting position with the head forward or in a lateral position, with the face downward at an angle sufficient to cause the fluid to escape from the nose. I can recall several cases in which the drainage of fluid was overlooked until the patients were sufficiently conscious to demonstrate its presence.

Depressed and *compound fractures* in themselves are important from the standpoint of infection, the cosmetic appearance, and possible changes in the underlying structures. The diagnosis of these skull injuries can be made by inspection and palpation of the wound and by *x*-ray examination. The extent of damage to the meninges and brain beneath may be indicated by the escape of cerebrospinal fluid or traumatized brain. If the dura, however, has not been torn, the diagnosis will depend upon the clinical course or upon inspection at operation.

CLASSIFICATION OF TRAUMATIC INTRACRANIAL LESIONS

Many outlines of intracranial lesions after skull injury have been suggested. One of the earlier classifications divided the cases into concussion, contusion and compression. Greater definition has been attempted in later classifications, but these are still not sufficiently inclusive. I suggest the following classification which separates those cases in which there is no escape of blood—namely, concussion and edema—from those with structural alterations in the form of hemorrhage. Hemorrhage alone is responsible for many of the signs presented by the patient. The variation in these signs is due to the location and quantity of blood. A large clot may produce

only signs of compression, while a microscopic extravasation may produce focal signs. Basing the diagnosis on a particular type of hemorrhage will stimulate a greater interest in the pathology of cerebral trauma, for it requires a knowledge of the pathology beyond such gross lesions as clots, contusions and lacerations.

I. Concussion.
II. Edema, prolonging the signs of concussion for a period ranging from several hours to several days.

From this point the classification is concerned with the type and location of bleeding.

III. Hemorrhage:
    1. Extradural hemorrhage, usually secondary to a tear of the middle meningeal artery or one of its branches, or of one of the sinuses of the dura.
    2. Acute subdural hematoma.
    3. Subarachnoid bleeding.
    4. Single or multiple large cortical clots.
    5. Multiple small intracerebral hemorrhages due to:
        (a) Fat emboli.
        (b) Extravasations in or around the brain stem, as in traumatic encephalitis.
        (c) Extravasation secondary to torsion of the vein of Galen, following a definite pattern as to type and distribution.
        (d) Venous extravasation into the cortex due to partial or complete thrombosis of the longitudinal sinus.

DIFFERENTIAL DIAGNOSIS

I. Concussion.—The term concussion should be reserved for cases in which there are no structural changes and where there is no extravasation of blood. Trotter in 1924[2] made the following pertinent statement: "I may say at once that I use the term concussion as I think it should be used in the strict classical sense to indicate an essentially transient state due to head injury which is of instantaneous onset, manifests widespread symptoms of a purely paralytic kind, does not as such comprise any evidence of structural cerebral injury and is always followed by amnesia for the actual moment of the accident." This definition permits a wide variation in signs, demanding only that they be transient. In the mild cases the whole period may last but a few minutes. In practice there must be some classification of the cases in which there is

prompt and complete return to normal after a few hours. The prompt recovery justifies the belief that there has been no structural alteration. Therefore, in spite of the fact that the symptoms do not immediately disappear, we prefer classifying this case as one of severe concussion. If the signs are prolonged over a period of several days and still clear up promptly, we assume that edema has ensued. The variable symptomatology will at times embrace one or more disturbances, suggesting a grave cerebral lesion, but if due to concussion alone the absence of other serious signs and the rapid shifting of those which are present will guide one to the correct diagnosis.

II. Edema.—The course in the patient with edema differs from that in the patient in whom there has been an extravasation of blood, in that the signs disappear rapidly because of the lack of structural changes. In the edema cases the pressure may not be altered, but depending upon its location the pressure may be subnormal or sufficiently increased to cause the usual signs of intracranial pressure, including changes in the optic disc.

III. Hemorrhage.—In the differential diagnosis of hemorrhage, the lesions can first be divided into the *large clot*, producing signs of compression, and the *small extravasations* extending into the meninges or brain substance, producing irritative signs.

THE LARGE BLOOD CLOT.—The *signs of compression*—severe headache, nausea and vomiting, slow pulse, and gradually increasing coma—have long been recognized. However, the interpretation of these signs is seldom simple. The patient in the early stage of a clot formation may be restless and the pulse may be rapid. He may become combative and be hustled off to a cell in a police station, or be placed in a strait-jacket in a hospital. Later he may be thought to be sleeping or under the influence of a sedative, while actually he is in coma. Because of this changing status, frequent neurologic examinations are necessary. This is true of a large clot in any portion of the brain, whether it be extradural, subdural or intracerebral.

The uncomplicated *extradural clot* presents the purest form of cerebral compression. Formerly these clots were con-

sidered to be secondary only to a tear in the middle meningeal artery or some of its branches. During the past few years a number have been found following tears in the lateral sinus, the blood escaping above or below the sinus and producing extradural clots overlying the posterior part of the cerebral hemispheres or the cerebellum. In those cases with hemorrhage in the posterior fossa one must depend entirely upon the signs of increased intracranial pressure. Such important signs as homolateral dilatation of the pupil and contralateral paralysis are absent.

Subdural clots are found after many lesions, such as fractures at the base, or tearing of the pial arteries, the veins or dural sinuses. If they are secondary to a torn sinus, a clot may form on each side of the sinus, producing a so-called "saddle" clot. Clots may be over any portion of the cortex of the cerebrum or cerebellum. Clots overlying the cerebral hemispheres may attain a large size before producing focal signs. On the contrary, a relatively small subdural clot overlying the cerebellum may produce a marked increase in intracranial pressure by interfering with the flow of cerebrospinal fluid from the fourth ventricle.

Intracerebral clots may be multiple and frequently result from the rupture of a pial vein on the cortex side, the overlying meninges preventing the escape of blood into the subarachnoid space. In some cases the only indication of the clot may be the gradually increasing intracranial pressure; in others, this may be associated with focal signs.

EXTRAVASATIONS INTO MENINGES OR BRAIN.—There is a large group of patients with serious head injuries in whom the pressure range is normal or below. It is to this group that I wish to call particular attention. There is extravasation of blood in these patients, the quantity of blood being much too small to cause through its bulk an increase in intracranial pressure, but the signs and symptoms are produced by the irritating effect of the blood.

The extravasated blood may be in the form of small *petechial hemorrhages*, as seen in fat embolism, where the lesions are scattered widely through the brain. The hemorrhages here are due to the plugging of small vessels by fat, followed by leakage.

A second type is represented by the group of cases in which there are numerous *extravasations in and about the brain stem*. The location of the hemorrhages in these patients is very similar to that of the inflammatory lesions found in lethargic encephalitis.

In a third group, small *venous extravasations* are seen in the area drained by the *internal cerebral and basilar veins*. The hemorrhages are believed to be caused by the blocking of the vein of Galen and the pattern of distribution is very definite.

Other patients in the low pressure irritative group are represented by the so-called *bloody cerebrospinal fluid type*. Here the signs—moderate elevation of temperature, pulse, and respiration, posterior cervical rigidity, positive Kernig, delirium, and restlessness—are due to the irritating effect of the blood upon the meninges. The symptoms may be very mild or sufficiently severe to cause death. The course depends upon the absorption or mechanical removal of the blood from the fluid. Absorption will suffice in mild cases, but if the progress of symptoms is unfavorable, fractional removal of the bloody fluid by lumbar puncture or decompression and drainage will be required.

The final group in the classification is the venous extravasation due to *partial or complete blocking of the posterior part of the longitudinal sinus*, causing dilatation and rupture of the cortical veins. The extravasations may be small and produce an irritative state, with severe convulsive seizures. The hemorrhages, however, may be sufficiently numerous and large enough to produce increased intracranial pressure. This lesion, therefore, may be of the low pressure irritative or high pressure compression type.

TREATMENT

It is hoped that the foregoing discussion will show the great difference in the intracranial pathology that accompanies the wide variation in signs and symptoms, demanding accurate diagnosis and treatment directed toward a particular lesion. In the very beginning, therefore, one must avoid routine measures. An orderly analysis of the problem at hand is the beginning of rational treatment.

Lacerations of the Scalp.—During the period of observation immediately following the accident, scalp lacerations should be treated. Immediate treatment is valuable in preventing loss of blood and the onset of infection. If the physician originally examining the patient will take care of the scalp lacerations properly, any necessary craniotomy may be done through or near such sutured lacerations within the first few days, without danger of infection.

Depressed Fractures.—In treating depressed fractures it is not sufficient to remove the bone fragments which are depressed and leave the patient with a skull defect unless this defect is well covered by the temporal or occipital muscles. In correcting the condition, therefore, one must aim to use the fragments so there will be no skull defect remaining. Posttraumatic neuroses are much more difficult to handle in patients in whom there is a pulsating skull defect. Insurance companies are generally unwilling to insure individuals, even years after, with such defects. Elevation and replacement of the fragments can best be accomplished by making a curved incision skirting the rim of the defect, and turning back a flap with the fragments attached to the under surface. After elevation of the fragments the extent of damage to the dura and cortex and the possible presence of blood must be determined; further procedures will depend upon the findings. The fact that the wound is a compound one is not sufficient justification for sacrificing all the detached fragments. If the fragments are allowed to remain, the wound must be watched for signs of infection, and a secondary operation can be undertaken if necessary.

Concussion.—The treatment in the patient with a concussion must be symptomatic and need not be detailed here. A word of warning, however, concerning the mental state is necessary. Concussion will produce a variety of changes, varying from sleep to delirium. Very young children will sleep for an hour or so and no concern need be felt if other serious signs are lacking. More commonly, especially in adults, there is a period of delirium, the management of which is most important. The confused, combative patient gives the impression of a more serious injury than the patient in

coma. The delirious state, however, is much more likely to pass off than is that of coma. Mild sedatives may be necessary, but one must avoid converting delirium into a comatose state by sedation. A minimum of restraint should be used because the level of consciousness may be such that restraint will increase restlessness.

Edema.—The second group under the heading of "Edema" is included in the classification so that one may be able to consider an intermediate stage between the transitory state of concussion and the structural lesions. The treatment here is also symptomatic. In this period the physician is still standing by, waiting for the development of signs, and he may use such diagnostic measures as lumbar puncture, intravenous hypertonic solutions, saline purge, and x-ray. It is fair to consider intravenous injection of hypertonic solutions with the diagnostic procedures at this stage for, if the condition is one of simple edema, there will be a period of improvement after its administration.

LUMBAR PUNCTURE.—There has been much discussion concerning the advisability of lumbar puncture in the diagnosis and treatment of cerebral injuries. A careful study of these discussions and a review of experiences in practice lead to the conclusion that the procedure is most useful or dangerous, depending upon the type of lesion in a given case. We must, therefore, outline a plan which will make it possible to decide where the value of the procedure ends and the danger begins.

Diagnostic Lumbar Puncture.—In diagnosis the puncture should be used only as an aid after hours or even days of observation with careful neurologic checking. By so doing, unnecessary or ill-advised punctures will be avoided. There is no reason, for example, to do a lumbar puncture in a patient with concussion signs if these signs are to disappear within a few hours, and there is every reason to avoid a puncture in a patient with a large intracranial clot. Upon the clinician, therefore, must rest the decision and his opinion must be based upon an understanding of the pathology in the individual case. It is absolutely necessary to consider separately its diagnostic and therapeutic value. So much informa-

tion can be obtained in an initial puncture, one may be justified in employing it in a doubtful case if a careful technic is employed. The following points should be observed in every case: careful manometric reading, retention by the bedside of a small sample of fluid, and the comparison in daylight of the fluid with water to determine tinting. If the data obtained indicate that there is increased intracranial pressure possibly due to the formation of a clot, the procedure has been ill-advised, but no harm may come of it. Further punctures, however, can only demonstrate its harm. On the other hand, if a bloody fluid is obtained in the patient in whom the clinical course has suggested the condition, the patient will probably be benefited temporarily by the withdrawal of the fluid, and further therapeutic punctures are indicated.

Therapeutic Lumbar Puncture.—The value of drainage of bloody cerebrospinal fluid has been recognized for more than thirty years. Dr. Cushing in 1907[3] advocated drainage through subtemporal decompression. The operation was extensively used for all types of skull injuries for a number of years, but I think it is fair to state that the early and repeated employment of lumbar puncture has to an extent replaced the cranial operation in an increasing number of cases during the last twenty-five years.

Large Blood Clots.—In the treatment of large clots, nothing can be accomplished except through their removal as soon as possible after the diagnosis is established. In choosing the time for operation, however, consideration must be given to the general condition of the patient. All clots, even fairly large extradural ones, do not reach a size incompatible with life before the bleeding ceases. Therefore, if the signs of compression are moderate and the patient's condition, because of concussion or shock, is not favorable for immediate operation, delay may add to the safety of the procedure.

Subarachnoid Hemorrhage.—The management of the patient with subarachnoid bleeding after the diagnosis has been established has to do chiefly with the state of restlessness, the control of pain, and the freeing of the cerebrospinal fluid of the irritating blood. Mild sedation is often required and can usually be accomplished by use of barbiturates. Morphine should be avoided because of its depressing effect. Anodynes

for the severe meningeal pain are often better than hypnotics. The blood in the cerebrospinal fluid must largely be absorbed. If the amount of blood is not too great, spontaneous absorption will occur rapidly and within a few days all signs of meningeal irritation will disappear. However, delay in absorption increases the meningeal irritation so that there is actual thickening of the meninges, which further interferes with the process of absorption. I have frequently found in patients who have grown progressively worse over a period of from five to seven days a very rich mixture of blood and cerebrospinal fluid on the first tap, and a marked diminution in the richness of blood in fluid obtained twenty-four or forty-eight hours later. If, in the established bloody cerebrospinal fluid case, the symptoms cannot be controlled by repeated lumbar punctures, subtemporal decompression for more adequate drainage should be employed. Intravenous hypertonic solutions are helpful in the treatment of these cases.

Petechial Hemorrhages.—In the treatment of cases with *multiple small hemorrhages*, measures must be directed toward the support of the patient until absorption of the hemorrhage and repair of the area surrounding it can take place. In the cases *secondary to fat emboli* the fat cannot be removed, but attention to the primary lesions, probably a fracture of a skeletal bone, is essential. The few patients who recover after this lesion do so, perhaps, because of the limited distribution of the emboli.

The treatment of the cases with *extravasation of blood in and about the brain stem* is also of the "stand-by-and-wait" type. Obviously the scattered hemorrhages can be removed only by absorption. If the hemorrhages are in certain areas, death will result within the first few days. Absorption and repair of the numerous damaged areas may require weeks, so that these patients often run a very tedious course, during which time there is slow but progressive improvement.

In the *vein of Galen* cases, absorption of the extravasation again is to be hoped for, and the process may take many weeks.

In the last group, *complete or partial thrombosis of the posterior part of the longitudinal sinus*, improvement may occur because of absorption of the clot or the establishment

of collateral circulation in partially obstructed cases. It is very unlikely that any procedure would help in the completely obstructed cases.

SUMMARY

An attempt is made in this paper to outline a plan of diagnosis and treatment of head injuries sufficiently severe to produce unconsciousness. Emphasis is placed upon the various types of cerebral lesions occurring after injury.

A classification based upon a variety of lesions previously described in the literature is offered.

Treatment varies in the individual case according to the diagnosis of the type of lesion present.

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SUICIDE FROM THE INGESTION OF MERCURY

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MERCURY, when taken with suicidal intent, is practically always ingested in the form of the bichloride salt (*corrosive sublimate*). The tablets offered for sale are usually characteristically colored and shaped to warn the careless drugtaker and, indeed, accidental poisoning is today quite rare. In a series of 135 consecutive cases of mercury poisoning studied by us the drug was taken by mistake in only seven cases, and in a few of these the "mistake" was dubious.

INCIDENCE

There is no doubt that the fashion or "vogue" in suicide, insofar as the method employed is concerned, has changed considerably through the years. Leaving aside "jumping, hanging, shooting and carbon monoxide," which have always headed the list of suicides, the type of ingested poison has had wide variation in popularity down the years. Carbolic acid was once quite popular but gave way to the opium derivatives. Then, and especially from 1920 to 1930, bichloride poisonings became very frequent. From 1930 to the present the latter has tapered off so that, fairly recently, Gonzales[1] reported suicidal mercury poisonings in New York City were superseded by such drugs as potassium cyanide, lysol, and the barbiturates. In our own clinic, mercury cases within the last five years have dropped to about one fifth of those seen around 1925. Probably the rather widespread laws requiring registration of the mercury purchaser as well as the cooperation of the public press in not specifically mentioning suicide drugs have acted as deterrents in this respect.

FATAL DOSE AND AFFECTING FACTORS

Three grains of mercury by ingestion has proved fatal. Such instances, however, are rare. On the other hand huge doses have been taken with recovery, but again such cases are exceptional. It cannot be said that the fatality rate always rises in direct proportion to the amount of drug taken. Other factors besides the actual dosage play a very important role. Thus the same dose of drug *in solution* has proved much more fatal than the ingestion of tablets as such. It is our experience and that of others that ingestion of the drug on an *empty stomach* is more serious than when taken after a full meal. The prior ingestion of *alcohol* has not uniformly caused a higher fatality rate. In the oft-quoted case of Armstrong,[2] 192 grains in alcoholic solution was followed by recovery.

Emesis Interval.—Any given dose of mercury may often prove fatal or nonfatal depending entirely on the emesis interval. By this we mean the estimated time from the ingestion of the drug until vomiting occurs or gastric lavage is done. This important factor is of undoubted prognostic value, provided we can elicit it with accuracy from the patient. Usually (but not always) the fatality rate is directly proportionate to the length of the emesis interval.

Fatal Period.—Death has occurred within half an hour after the ingestion of mercury, but such cases are exceedingly rare. Fatalities within a few hours due to shock have been reported. We have seen no such cases in our clinic. Of the fatal uremic cases, many patients die within five days, but occasionally they may live for weeks.

PATHOGENIC ACTION OF MERCURY

Ingested mercury produces *local inflammatory changes* wherever it contacts the mucous membrane. Lesions, then, may extend only part way down the alimentary canal, or, on the other hand, one may see extensive damage from the mouth to the anus. Of graver import, however, is the further action of the drug. With surprising rapidity mercury is absorbed from the stomach and intestines into the circulation. From here part of it is re-excreted back into the alimentary canal, especially to the stomach and colon. Rather large quantities of mercury have been shown to pass through the

liver into the bile, and thus add additional insult to the intestinal tract.

By far the most pernicious and fatal action of the drug is its passage through the kidneys with the *nephrosis* thereby produced. The great majority of mercury deaths are "kidney deaths." The extreme rapidity with which mercury may pass from the stomach to cause irrevocable renal damage should always be borne in mind. When we realize that a definite nephrosis was found in animals five minutes after the introduction of mercury into their stomachs,[3] we will become wary of too sanguine a prognosis or of expecting too much from any mode of therapy in some of our human cases.

Finally, the combination of persistent vomiting and diarrhea plus a nephrosis induces at times a definite *lowering of the blood chlorides* as well as occasionally causing a well marked *acidosis*—all facts to remember in therapy.

PATHOLOGY

Only essential postmortem changes can be mentioned here. Damage to the *alimentary tract* and to the *kidneys* overshadows all else.

All degrees of gingivitis, glossitis, pharyngitis, gastritis, enteritis, and especially colitis, are to be seen. Lesions may be simple hyperemic, hemorrhagic, ulcerative, or even gangrenous. Perforations are possible but rare. The kidneys present a typical nephrosis with swelling and necrosis of the tubular epithelium. Calcium deposits may be seen but are not specific for mercury poisoning. Slight glomerular changes are a secondary phenomenon. The liver may show some fatty and parenchymatous lesions and the myocardium may present degenerative changes.

SYMPTOMS

Mercury will produce symptoms (1) by its effects on the circulatory system, (2) by its corrosive action along the gastro-intestinal tract, and (3) by it effects on the kidneys.

Circulatory System.—While in 135 cases seen by us *shock* has not been present to any appreciable degree, others have observed and reported it. It apparently may come on early after the poisoning, or it may be seen late in the course of an ulcero-hemorrhagic colitis.

Typical symptoms of pallor, weak and rapid pulse, low blood-pressure, subnormal temperature, syncope and collapse are present.

Gastro-intestinal Tract.—*Vomiting* or *retching* is one of the first symptoms and occurs very frequently within the first five to fifteen minutes. Occasionally we have been surprised at the ingestion of comparatively large amounts of mercury with no symptoms whatsoever for from thirty to sixty minutes or longer. Usually, soon after ingestion, patients complain of a *burning, metallic taste* in the mouth, with *thirst* and *soreness in the pharynx*. *Abdominal pain* soon is felt. The *gingivitis, glossitis, stomatitis* and *pharyngitis*, when present, may be of all degrees from simple reddening of the mucous membranes with salivation to most horrible pictures of dark, foul smelling, membranous, bleeding, ulcerated or sloughing necrosis. Marked *swelling* may occur and, very rarely, *asphyxia* has been reported. I have seen a glossitis with such swelling that the tongue protruded far outside of the mouth for days. (The patient, incidentally, recovered.) Vomiting is usually, but not necessarily, persistent and the vomitus is often bloody. The abdominal pain may gradually get worse and severe *purging* with liquid and later bloody stools may come on. There is usually associated *tenesmus* and *rectal pain*. Sloughing colonic membrane has been seen at times in the stools.

Kidneys.—Many of our mild cases showed practically no demonstrable kidney pathology. In more severe cases, however, *oliguria* followed often by *anuria* sets in at intervals varying from one to three days. In cases going on to suppression, the urine shows marked *albuminuria, cylindruria* and especially *hematuria*. *Pain in the loin region* sometimes occurs. With the advent of oliguria—and certainly after anuria—the nitrogenous elements in the blood quickly rise. A fatal case lasting several days might easily have a blood urea in the hundreds of milligrams, with a correspondingly high creatinine.

Uremia contributes its element of nausea and vomiting, and there may be *drowsiness, hiccup, twitching* of the muscles and, finally, *coma*. In one of our cases we observed late *purpura, bleeding* and *thrombocytopenia*. Convulsions are

but seldom seen. Practically never is there any rise in blood pressure and, despite the anuria, edema does not occur. Symptoms secondary to *lowered blood chlorides* and even those of a moderately severe *acidosis* may well be hidden in the general clinical picture. Hence routine tests should be made for these important and remediable complications.

PROGNOSIS

While in such cases prognosis should always be guarded, experience has nevertheless shown that certain factors enable us to predict with some degree of accuracy the trend of the case. Careful evaluation of all the factors set down under "Fatal Dose" will be helpful. Thus if 5 grains or less of the drug are taken, the outcome almost certainly will be good. If the emesis interval is definitely known to be less than one-half hour the prognosis is usually good, although there are exceptions. In our experience vomiting after mercury ingestion is often incomplete and ineffectual. Hence the length of time until actual *therapeutic lavage* is of prime importance. Cases with marked gastro-enteritis or definite nephrosis before coming to treatment naturally fall into a very serious and critical group.

Goldblatt,[4] Porter and Simons,[5] and others, have stressed the *early prognostic leukocytosis:* Before any marked degree of gastro-enteritis or nephrosis is seen, certain patients will show a leukocytosis of 20,000 or more, and such cases almost always prove serious. In our experience this statement usually holds true although there are undoubted exceptions. Any case with early uremia is certainly a critical one, but many case reports hearten the therapist in that occasional recoveries occur after a long suppression. McNally[6] has declared that "if the nonprotein nitrogen continues to increase after the fourth day, the prognosis is very grave" and lately[7] "if the nonprotein nitrogen begins to go down after the seventh day, you may be certain that the patient will recover."

TREATMENT

Emergency Treatment.—Remember that this initial treatment may be almost 100 per cent more effectual in saving life than any measure you will use later. If you are

acquainted of the case by telephone, instruct the relatives or attendants *at once over the telephone* as to correct first treatment. (Even if carried out only partially by the time you arrive on the scene, a great amount of mercury absorption may have been prevented.)

Instructions for Initial Treatment.—Allow or force the patient to drink 1 pint of *milk.* (Skimmed milk theoretically is better as fats dissolve mercury salts, aiding absorption.[6] However, do not search for skimmed milk when whole milk is handy.) If there is no milk give a pint of water. Follow the ingestion of the milk (or water) with ingestion of 2 or 3 whole raw eggs. (It is not necessary to separate the whites.) After only a few minutes induce vomiting with lukewarm salt water, mustard water, etc. On the arrival of the physician at the scene repeat the whole procedure up to the point of initiating vomiting. Then give a thorough *gastric lavage.* Sollman, Barlow and Biskind[8] have shown that the initial introduction of milk (or water) tends to keep the mercury tablets from "cementing" to the stomach wall, which might occur if the eggs are given first. Moreover, it renders the precipitated albuminate of mercury fine enough to pass through the gastric tube. Several writers advise against too much egg-white as they believe it favors the resolution of the mercuric compound. (The value of *intravenous antidotes* will be considered later.)

Should the rare phenomenon of *shock* be present hot blankets should be applied and the subcutaneous injection of *coramine,* 1 cc., or even *adrenalin chloride* (1:1000), 15 minims, must be considered. *Morphine,* later on, may be valuable, and if available a *plasma transfusion* would be ideal.

A word of warning should be given here. It must be remembered that the patient was bent on suicide, and occasionally even nausea and vomiting will not deter him from a sudden further attempt at self-destruction. Hence, *strict instruction against leaving the patient unattended must be given.* Also, at times, the cunning patient denies "really swallowing any poison" in the hope that treatment will be omitted. Contrariwise a psychically unstable or love-lorn individual may, to gain his ends, falsely declare he has taken

mercury. The treatment in all such cases is the prompt application—*by force* if necessary—of the measures outlined above. Decide later from chemical analysis of the vomitus and urine whether or not mercury has been taken.

Further Therapy After Emergency Treatment.—Since any patient who has ingested mercury may, despite proper emergency therapy, be a critically ill person within twenty-four hours, *hospitalization* is practically a necessity. Only here can the patient have the continuous observation, proper laboratory studies and the rather strenuous treatment that is required.

Aside from antidotal measures, we believe most therapists today adhere in the main to principles advocated long ago by Lambert and Patterson.[9] These included immediate, intensive and prolonged efforts to prevent as far as possible *further absorption of re-excreted mercury*. In addition, the forcing of large quantities of *alkaline drinks* and *parenteral fluids* was advocated in order to dilute toxins and promote their elimination by all possible channels. The value of a large intake of fluids in such cases soon was corroborated by many[10, 11, 12] and such practice lines up well with modern concepts of treatment in nephrotic cases without edema and with threatened or actual anuria.[13, 14] Added to these general measures, we today stress the importance of frequent *routine checks of the blood* for hypochloremia and acidosis, and we make every effort therapeutically to adjust such figures to normal.

The practical application of the foregoing principles is specifically enumerated in the following:

ROUTINE TREATMENT

Fluids by Mouth.—Eight ounces of a *mildly alkaline diuretic* are given every four hours. Such drink might be the rather pleasant *"potus imperialis"*:

℞ Potassium bitartrate	....	1 dram
Sugar	.....	1 dram
Lactose	......	½ ounce
Lemon juice	....	1 ounce
Water	q.s. ad	1 pint

However, we most often have used *potassium citrate* and *potassium acetate*, 15 grains each to water, 8 ounces. Alternating with this drink, 8 ounces of *milk* are given every four hours.

Such fluids are urged on the patient irrespective of vomiting, and in almost all cases they will quiet rather than aggravate the vomiting. At all events, if early in the treatment they exert an emetic effect, a desirable lavage action is thus obtained.

Gastric Lavage.—This should be performed gently with plain water every eight hours until several lavage specimens remain negative for mercury. The procedure must be omitted if definite hematemesis is present. (McNally[7] uses 1 quart of water containing 8 grains of calcium sulfide and lavages every four hours.)

Colonic Irrigation.—This is given every eight hours until several washings remain negative for mercury. A mild soda solution may be used. In very severe cases of ulcero-hemorrhagic colitis the irrigations are inadvisable.

Hypodermoclysis and Proctoclysis.—While used extensively in the past, these have given way largely to more effectual parenteral therapy.

Hot packs, recommended by Lambert and Patterson, are today frowned upon by most therapists.

Intravenous Fluids.—In these nonedematous patients a daily intake of 3500 to 4000 cc. is desirable. Where much fluid is lost by vomiting and diarrhea, an even higher intake is required and intravenous injections are a practical necessity. *Ten per cent glucose in saline* is given in 500 cc. quantities to make up the oral deficit. The rate of injection must be very slow, or preferably, given as an intravenous drip. The blood chlorides and the plasma CO_2 combining power should be estimated daily for the first few days or longer if the patient is still critically ill. Lowered blood chlorides must be raised by *1 per cent sodium chloride intravenously* when necessary, although *normal saline is often sufficient.* If the blood CO_2 is much below 40 volumes per cent, intravenous injections of *5 per cent sodium bicarbonate* (500 cc.) must be given at intervals until the acidosis is relieved.

Blood Transfusion.—This is specifically indicated only in cases associated with hemorrhage or shock. Where there is shock with dehydration or hemoconcentration and no hemorrhage, *plasma transfusions* are especially indicated.

Operative Procedures.—*Colostomy* now has but few proponents and *kidney decapsulation* in any anuria is today in rather wide disfavor.[13] *Psychiatric examination* after recovery may be more permanently life-saving than any other measure.

INTRAVENOUS ANTIDOTES

Remembering the extreme rapidity with which mercury may be absorbed and set up grave kidney lesions, the possibility of an intravenous antidote which will neutralize the absorbed mercury in situ has always intrigued medical investigators. The most widely discussed of such drugs are *sodium thiosulfate,* advanced in 1923 by McBride and Dennie[15] and *sodium formaldehyde sulfoxylate,* offered in 1933 by Rosenthal.[16]

Medical literature is well filled with reports for and against these antidotes. Indeed, one feels that the attention of the therapist is often so confined to antidotal measures that

proper and adequate treatment of the damage *already done*
by the mercury is slighted. Within the last year a growing
feeling against the efficacy of these drugs has culminated, so
to speak, in two independent reports—one based on experi-
mental evidence and the other on clinical experience—and
their conclusions are most discouraging. Muir, Stenhouse
and Becker[17] failed to protect rabbits from mercury (or
arsenic) when these sulfur compounds were given *after* the
poisoning. It is interesting that when given *before* the
poisoning, a protective effect could be demonstrated. Monte
and Hull,[18] in a series of forty cases treated with sodium for-
maldehyde sulfoxylate, found on careful analysis that the
mortality was actually higher than in 278 prior cases treated
by other methods.

In a series of 144 consecutive cases studied in our clinic,
all patients were given treatment based in general on the prin-
ciples herein outlined and, in addition, all received intra-
venous injections of sodium thiosulfate or, in recent years,
sodium formaldehyde sulfoxylate. This procedure was part
of the *emergency* treatment in the accident department.
Omitting a number of dubious cases, the series totals 135
cases of mercury poisoning. There were eighteen fatalities,
giving a mortality of 13.5 per cent. Superficially such a low
rate in a goodly series of cases is pleasing, but it must be
remembered that on breaking down the cases as to severity,
there is naturally a great increase in fatality rate among
certain groups. In a general way we can say that the average
dose taken and the average emesis or lavage interval are com-
parable with most reported series of cases and that patients
developing a marked nephrosis were comparatively few.
Those still fired with enthusiasm for the intravenous sulfur
compounds may still argue that the prompt emergency use
of the drug in the accident department "protected" the renal
cells from further continuous insult by re-excreted mercury
or mercury from the liver, and hence comparatively few
"toxic" cases resulted. At all events, as a single emergency
dose of the drug in our opinion is comparatively innocuous,
we shall continue its use in the accident department for the
present. Leaving aside initial antidotal measures we believe

that judicious treatment of the *effects* of mercury cannot be too strongly stressed.

CONCLUSIONS

A great many cases of mercury poisoning are so mild that we realize the patients would have recovered without any treatment. Conversely, we appreciate in retrospect that in certain fatal cases the patients would have died despite any treatment. Between these are the borderline cases for which we must fight and in which occasionally the battle is won. Since any case of incipient mercury poisoning may develop into this borderline class, *every patient* should receive the full, complete and strenuous therapy which has been outlined.

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SUICIDE FROM THE INGESTION OF VERONAL OR OTHER BARBITURIC ACID DERIVATIVES

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BARBITAL was introduced by Fisher and Mering in 1903 under the name of Veronal. Since then a large number of cases of poisoning have been reported, some accidental, most of them suicides or attempts at suicide. The public is using a larger quantity of this drug than is realized, and, while first orders are sold on "prescription only" in most states, "refills" can be obtained indefinitely on the original prescription. Addiction is, therefore, not uncommon.

The Board of Trustees of the Council on Pharmacy and Chemistry of the American Medical Association at its annual meeting in March, 1938 requested the Council to investigate and report on the promiscuous use of the barbiturates. Hambourger[1] made this report in 1939, and he showed that sales of barbituric acid and its derivatives in the United States, with and without medical supervision, amounted to 1,215,-000,000 grains in 1936.

The United States Bureau of Census reports that within the five-year period 1932–1936 there were 634 successful suicides by all barbiturates, or 2.2 per cent of suicides by all drugs and poisons, gaseous as well as liquids and solids. Barbiturate suicides constituted 0.66 per cent of suicides by all means. Hambourger likewise shows that the data for this five-year period indicate a definite increase in recent years, in the incidence of suicide by barbiturates, whereas the incidence of suicides by liquid and solid poisons, and of total suicides, have both slightly declined. He cites a report from the Metropolitan Life Insurance Company which showed that 6 per cent of all suicides by drugs, of their policy holders,

were due to barbiturates during the years 1935–1937. During the same period 18 per cent of accidental deaths were caused by barbiturates. From the foregoing it appears that uniform Federal legislation is needed to cover all States and to prevent "refilling" of prescriptions indefinitely.

Veronal (barbital, barbitone, diethylmalonylurea or diethylbarbituric acid), $(C_2H_5)_2:C:(CONH)_2CO$, is one of a large number of closely related barbituric acid derivatives. It is a white crystalline powder, slightly acid in reaction, bitter to taste, and without odor; 1 gm. is soluble in 150 cc. of water and 14 cc. of alcohol at 25° C. It melts between 187° and 190° C. It is absorbed in the intestine, not in the stomach, and is eliminated by the kidney as veronal (50 to 90 per cent) while the rest is oxidized in the system. The rate of excretion is slow and, when the drug is taken in large doses, excretion is at its height probably on the second day but continues through the fourth day. In doubtful cases of poisoning, chemical examination of the urine is important for diagnosis. In therapeutic dosage 0.5 gm. (7½ grains) veronal induces sleep. Small doses allay nervousness and restlessness, increase in dosage is followed by analgesia, amnesia and anesthesia.

Pharmacologic Action of Veronal.—After its ingestion, veronal affects bodily functions as follows: The sensitiveness of the stomach is decreased, which action is probably central, motor activity of smooth muscle is depressed, and the tone and activity of the entire intestinal tract is diminished. Anesthetic but not hypnotic doses cause a fall in blood pressure. Like the other barbiturates, veronal acts directly on vessel walls and causes vasodilatation of the peripheral circulation, and at times cyanosis. Rarely, edema may follow due to change of capillary permeability. The respiratory center is depressed and respirations may become shallow and rapid or occasionally slow and deep. The nervous system is generally affected, reflexes are depressed, as are also the cardiac vagus endings.

Koppanyi[2] claims that veronal is evenly distributed throughout the central nervous system irrespective of whether the dose is large or small. Keeser and Keeser[3] state that larger concentrations of the drug were found by them in the

midbrain and thalamus. Blood chemistry studies show that there is an increase in blood sugar and a decrease in blood calcium and phosphorus.   Many side actions have been noted.[4]

Poisoning by Veronal and Other Barbiturates.—One must distinguish between the *toxic reactions* which occur when a small dose of the drug is taken by someone with an idiosyncrasy, and *acute poisoning* caused by an overdose of the drug in a normal person; or, overdosage in an addict where the cumulative factor plays a role. An *overdose* producing death has been reported as ranging from a minimum of 10 grains (0.65 gm.) to a dose of 150 grains (10 gm.) which is nearly always fatal. Chang and Tainter[5] report a case with recovery after the ingestion of 270 grains (18 gm.). The average dose for suicidal purpose is 50 grains (3.3 gm.). The *lethal* dose varies with the type of barbiturate used. To avoid the reactions that sometimes occur in persons who are drug sensitive, Poole[6] advocates a skin test before starting therapy with barbiturates.

According to Koppanyi[7] the barbiturates are classified as either *long-* or *short-acting,* veronal falling into the former group. This classification is on the basis that in the case of long-acting drugs there is less concentration in the brain, with less accumulation in the medulla and more in the cerebrum than in the shorter acting drugs, and furthermore, the concentration in the brain remains for a longer period of time in this group.

Clinically, the *toxic reactions* to the whole group of drugs are about the same: somnolence, stupor, pupils dilated at first but in stupor often hippus, relaxation of all muscles, decreased reflexes, hypotension, tachycardia, and coma. At first, the temperature is subnormal, but this is followed by fever. In addition to these signs selective reactions may or may not occur, for example, dermatitis, either urticarial or a scarlatiniform erythema. Neuritis occurs at times. Anemia is comparatively common and neutropenia has been reported.[8] Incontinence of bowel and bladder, retention of urine, conjunctivitis, diarrhea and cardiac arrythmias have all been observed. An interesting psychologic symptom of amytal poisoning called "automatism" was noticed by Richards.[9]

Autopsy Findings.—In a case of acute poisoning nothing characteristic may be seen grossly. When life has been prolonged for a number of days there is cyanosis, engorgement and congestion of the viscera, with hyperemia and edema of the meninges and perivascular hemorrhage. The heart is dilated and the lungs are congested and edematous and frequently show patches of pneumonia as a result of prolonged coma. Atelectasis and pulmonary abscess occur. The kidneys show degeneration of the convoluted tubular epithelium and petechial hemorrhages in the pelvis. The liver shows evidence of fatty degeneration. Capillary congestion and hemorrhage may be found in the soft palate, the submucosa of the stomach, and the small intestines and mesenteric lymph nodes. The brain, liver, kidneys and spleen contain large amounts of the drug, and in fatal cases these organs should be saved for the toxicologist.

Symptoms.—Large doses of veronal cause depression of the central nervous system. Dizziness and headache are common complaints. Nausea occurs, and sometimes vomiting. Muscular weakness, with twitching and later ataxia, mental confusion and inarticulate speech is the sequence in poisoning, to be followed by sleep and coma. Shallow respirations (sometimes noisy), diplopia and alternate contraction and dilatation of the pupil (hippus) are features. Later there is a distinct fall of blood pressure due to paralysis of the peripheral blood vessels. Cyanosis may be pronounced, skin rash may be present.

The patient's coma may be either a profound stupor or be associated with restlessness and tonic spasms. This coma usually deepens; the patient may recover after an interval of several days, or death may occur from respiratory paralysis, vasomotor collapse, uremia or bronchopneumonia.

Diagnosis.—A *history of ingestion* of the drug is an extremely valuable aid in diagnosis. When no such history is obtained and one sees a patient in coma whose reflexes and pupillary reactions, though obtunded, are obtained, and who is cyanotic, has a low blood pressure and a subnormal temperature, barbiturate poisoning must be suspected. *Hippus*, when present with the other features, is diagnostic. Any of the accepted *chemical tests* of Koppanyi, Murphy and Krop.[1n]

Brundage and Gruber,[11] or Delmonico,[12] can be employed for extraction of the drug from the blood, urine or gastric contents. The presence of the chemical in body fluids or excretions is confirmatory of poisoning.

Barbiturate coma must be differentiated from *epidemic encephalitis, opium poisoning* and *uremia*. In encephalitis, the acute onset with coryza, fever and leukocytosis, and the external or internal ophthalmoplegia, should excite suspicion of an acute infection. The spinal fluid may be normal, but it usually shows an increase in globulin and cells and an increase in sugar. In opium poisoning the face is flushed, the pupil pin-point in size, and the respirations more depressed than in veronal poisoning, at times being as low as 4 to 7 per minute. Cheyne-Stokes respiration is seen in both conditions but is more persistent in opium poisoning. The presence of punctate cutaneous scars (needle sticks) should arouse suspicion of opium poison. In uremia, urine studies that show albuminuria, cylindruria and hematuria, and blood chemistry changes are valuable aids in differential diagnosis and albuminuric retinitis is specifically diagnostic. The prognosis in barbiturate poisoning should always be guarded.

TREATMENT

Supportive Therapy and Elimination of the Poison. —The physician is usually called when the patient is in coma. Therapy is directed to general systemic support, efforts to get rid of the poison, and efforts to stimulate the nerve centers.

The stomach should be *evacuated* even when the drug was taken many hours before. This should be done with the head lowered to prevent aspiration, and if trismus is present, a tube should be passed through the nose. *Lavage* may be done with 1:5000 potassium permanganate solution. Two to 3 ounces of sodium phosphate can be left in the stomach and enemas may also be required. *Postural drainage* should be continuous, with the head of the patient turned to the side and suction employed at periodic intervals for the removal of secretions from the pharynx.

If *cyanosis* is pronounced, oxygen can be administered through a nasal catheter, and when respirations are shallow, a few whiffs of carbon dioxide may be given at half-hour intervals

until the respiratory rate is improved. *Tube feeding* of a high caloric liquid diet should be carried out at four-hour periods with a preliminary lavage with sodium bicarbonate solution before each feeding. To overcome anuria, stimulate the elimination of the drug, and to maintain a normal water balance, *intravenous glucose* (5 or 10 per cent in physiologic salt solution) should be given. *Catheterization* may be required at four- to eight-hour intervals. *External heat* may be necessary for the maintenance of a normal body temperature, but great care should be exercised to prevent burns.

Stimulants.—For antidotal purposes Maloney[13] claims *picrotoxin* should be the drug of choice; Maloney and Tatum[14] advise vigorous antidoting following ingestion of short-acting barbiturates and smaller but more frequent doses of antidote in the longer-acting drugs. Sufficient evidence has accumulated to recommend picrotoxin as the most powerful stimulant in any case of severe barbiturate poisoning. The drug may be given intravenously, subcutaneously or intramuscularly. The amount and method of administration depend on the clinical symptoms exhibited and the quantity and type of barbiturate ingested. In coma, it may be given intravenously in doses of 3 to 12 mg. as frequently as every half hour. The drug has a well marked latent period, but when it acts, its effects extend from one-half to three hours and its action is to bring back the excitement of the motor cortex which has been abolished by the veronal. It is used as a 0.3 per cent solution and each cc. contains 3 mg. Totals of over 600 mg. have been used in the treatment of barbiturate coma. Twitchings of facial muscles are a sign that optimum effect of the drug is being obtained. Complete awakening is undesirable, as this may lead to convulsions. Picrotoxin treatment should not be undertaken without having close at hand a soluble barbiturate for intravenous use in case convulsions develop. Retching, vomiting, restoration of cough, improvement in respirations and swallowing and the production of spontaneous movements are all signs that one may change to the use of the drug intramuscularly, instead of intravenously, and at hourly intervals. If coma deepens, more frequent intravenous medication may be started again.

Metrazol is likewise a powerful stimulant, especially of the respiratory center, but its action is not so prolonged (lasting

only ten to thirty minutes) as the action of picrotoxin. It has no latent period and its maximum effect is almost immediate. One to 2 cc. (1½–3 grains) of a 10 per cent solution (1 cc. equals 0.1 gm.), may be given intravenously every fifteen minutes until the desired effect is obtained, then intramuscular medication may be employed at half-hour intervals. Alternating metrazol and picrotoxin is a plan of therapy which would result in a marked and prompt stimulation of the respiratory center by metrazol, which action would be increased and prolonged by the addition of the picrotoxin. *Nikethamide* (pyridine-beta-carboxylic acid diethylamide), a derivative of nicotinic acid, is a quick-acting analeptic, vasopressor and respiratory stimulant, but is inferior to picrotoxin and metrazol as an analeptic in severe barbital poisoning. It has been used in the treatment of poisoning by barbiturates in 25 per cent aqueous solution, 2 cc. equalling 0.5 gm. of the drug.

With collapse of circulation and a fall of blood pressure below 80 mm. of mercury systolic, *adrenalin* and *ephedrine* are indicated. *Digitalis* and *strophanthin-K* have been advised, but in my opinion, these drugs are not indicated in this type of failure.

Lumbar puncture should be done in all cases of deep coma. Cerebral edema is a frequent and serious complication, and if spinal fluid pressure is elevated, its reduction is imperative. Intravenous injection of sucrose, or sorbitol, will reduce intracranial pressure. Constant *nursing care* is required for the prevention of pulmonary complications and decubitus ulcers.

DISCUSSION AND CONCLUSIONS

Suicide from the ingestion of veronal and other barbituric acid derivatives is on the increase, the mortality rate being over 20 per cent. To prevent the public from using such large quantities of these drugs, Federal legislation should be enacted to limit the number of "refills" obtainable on an original order.

Poisoning may occur from the long- or short-acting barbiturates. Veronal belongs to the former group. The reported lethal dose of veronal has ranged from a minimum of 10 grains (0.6 gm.) to a dose of 150 grains (10 gm.) or more. The average case of overdosage is 50 grains (3.3 gm.).

The history of ingestion is a very valuable aid in diagnosis of barbiturate poisoning. Chemical tests may be employed for the extraction of the drug from tissue, urine or gastric juice.

From the accumulated evidence, treatment consists in the use of picrotoxin alone, or the alternation of picrotoxin with metrazol. The prognosis should be guarded.

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SUICIDE AND HOMICIDE BY VIOLENCE

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Suicide and homicide by violence, such as shooting, hanging, jumping, cutting, stabbing, assault and strangulation, are presented here with the aim of apprising the general practitioner of the essential features of these deaths and of the problems that may confront him on encountering a suicide or homicide.

Incidence and Method of Suicide and Homicide.— Suicide.—The general rate of suicide per 100,000 population in the United States during 1938 was 15.1, which is slightly above the average of 13.8 for twelve countries whose annual reports are available for 1938. Countries with the highest rates were Switzerland, Denmark, Finland, the United States, England and Wales, New Zealand, Australia and Scotland. Germany, Japan, Austria and Czechoslovakia normally have rates among the highest. Suicide rates in foreign-born groups in this country are usually appreciably higher than are those for the corresponding native country.

In the United States, suicide rates are highest in states on the west coast and lowest in states comprising the deep south. They are more frequent in cities than in country districts. More suicides are committed in spring and early summer than at other times of year. The vast majority of suicides are committed between 6:00 P.M. and 6:00 A.M. Rates are high among physicians, dentists and lawyers, and low among clergymen and teachers; they are high among the divorced, single and widowed, and low among the married. The insane, alcoholics and drug addicts commit most of the suicides. Rates are high for the rich and the poorest of the poor. Two or three males commit suicide to one female in almost all countries. The rate is considerably higher in whites than in Negroes, and

TABLE 1
INCIDENCE OF SUICIDE AND HOMICIDE

	Suicide						Homicide					
	Per 100,000 Population					Per 1000 Deaths	Per 100,000 Population					Per 1000 Deaths
	Total	Male	Female	White	Colored	Total	Total	Male	Female	White	Colored*	Total
Maryland, June 1, 1939 to May 31, 1940 — State	14.3	21.6	7	16.3	4.5	12.1	6.3	10.3	2.2	2.1	26.4	5.3
Counties	12.6	19.3	5.7	13.6	4.3	11.8	5.5	8.7	2.3	2.0	25.0	5.2
Baltimore	16.4	24.1	8.3	19	4.7	12.5	7.3	12.3	2.1	2.2	27.6	5.5
New York City, 1935	16.9	24 5	9.2	..	..	16.1	5.6	..	..	..	..	5.3
Buenos Aires, 1938	18.5	26	11.1	..	..	15.1	15.6	13.4	18.0	..	..	13.0

*Seventy-two per cent of homicides occurred among Negroes who comprised 17 per cent of the population.

TABLE 2

METHOD OF SUICIDE

| | Maryland, June 1, 1939 to May 31, 1940 | | | | | | | | | New York City, 1935 | | |
| | State | | | Baltimore | | | Counties | | | | | |
	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total	Male	Female
Shooting.................	37*	44	15	34	43	8	41	45	26	11	15	0.6
Illuminating gas.........	20	16	30	29	23	47	8	8	7	34	31	41
Hanging.................	16	14	21	11	10	17	20	19	26	20	24	12
Poisoning...............	10	8	.17	11	10	17	9	7	18	12	10	18
Motor exhaust...........	6	8	2	3	3	0	10	12	4	12	10	18
Jumping.................	5	5	8	7	8	6	3	1	11	1	1	0.3
Drowning...............	3	2	5	1	0	5	5	5	4	17	14	26
Cutting.................	2	2	2	1	2	0	3	2	4	2	2	1
Fire....................	0.3	0.5	0	0.7	1	0	0	0	0	3	4	1
Total number of suicides......	258	195	63	140	104	36	118	91	27	0.1	0.1	0
Suicides in whites........	244	185	59	132	97	35	112	88	24	1210	881	329
Suicides in Negroes........	14	10	4	8	7	1	6	3	3	..	..	..

* Per cent.

Negroes have the lowest rate of the colored races. Suicide increases with age per 100,000 population of age group. It is highest between 65 and 75 years but more suicides are committed in younger age groups with greater population. Suicide is rare below fifteen years of age.

In the late 1920's the most common method, by far, of committing suicide was by shooting; then came hanging, poisoning, and asphyxia by gas. In the male, the frequency of the various methods was, in order named, shooting, hanging, gas and poisoning; in the female it was poisoning, gas, shooting and hanging.

HOMICIDE.—The incidence of homicide per 100,000 population in the United States was 6.8 for 1938. This was the highest figure for all countries with available reports. Only Finland approached this with a rate of 5.2. The average rate for ten other countries was 0.8. The rate for England and Wales was 0.4, for Scotland, Denmark and the Netherlands 0.5, and for Canada 1.1.

With the exception of several western states, the homicide rate is highest in states with the highest percentage of Negro population. It is most common between the ages of twenty and thirty, far more common in the male than the female, and more common in cities than in country districts. As a rule, homicide is highest in communities or regions where punishment for the crime is slight and public opinion is least aroused by the act. One of the most important factors in the rate of homicide is the availability of lethal weapons. Other things being equal, a high rate of illiteracy favors a high rate of homicide. This is best explained, perhaps, by the usual lack of individual and communal responsibilities in illiterates. The effect of imbibition of alcoholic drinks is notorious for its predisposition to commit homicide.

Determination of Death.—Homicide or suicide is not a fact until death has been established. Practicing physicians are called to pronounce dead the majority of these individuals. Usually, the medical examiner or coroner is called by the police after a neighborhood or hospital physician has established death. The establishment of death in the majority of cases presents little or no difficulty. There are occasional cases, however, in which the person pronounced dead has manifested

TABLE 3
METHOD OF HOMICIDE

Maryland, June 1, 1939 to May 31, 1940

	State			Baltimore			Counties			New York City, 1935
	Total	Male	Female	Total	Male	Female	Total	Male	Female	Total
Shooting..............	60*	57	70	60	58	67	60	56	72	49
Stabbing.............	24	23	20	26	26	22	21	20	18	27
Assault..............	16	19	10	14	15	11	19	24	9	22
Strangulation.......	0	0	0	0	0	0	0	0	0	3
Total number of homicides .	114	94	20	62	53	9	52	41	11	399
Homicides in whites.......	32	26	6	15	11	4	17	15	2	
Homicides in Negroes......	82	68	14	47	42	5	35	26	9	
Wounds in chest......	45*	..	..	52	..	..	37			
Wounds in head......	28	..	..	21	..	..	37			
Wounds in abdomen......	14	..	..	13	..	..	15			
Wounds in neck......	9	..	..	10	..	..	8			

* Per cent.

life after various intervals. Consequently, one should make frequent auscultations of the heart and observations of the chest for respiratory movements *for a period of five minutes or more*. If cessation of circulatory and respiratory function can be established for a period of five minutes, death has occurred.

The most obvious *signs of death* are rigor, lividity and coldness. No one sign is entirely reliable, although rigor and lividity, when accurately determined, constitute irrefutable evidence of death. Rigor mortis and lividity may be present in warm bodies and absent in cold bodies. Rigor mortis is best differentiated from spasticity of muscles by noting the absence of return to original position of joint after flexing in the former. Lividity is due to gravitation of blood into capillaries of skin over dependent portions of body. Therefore, dependent portions of the body should be examined to establish this sign. Occasionally, circumscribed small hemorrhages (Tardieu spots) form in the dependent parts, especially in hangings and in bodies lying face down. Such hemorrhages in the mucous membrane of the nasal cavity may rupture externally and simulate antemortem bleeding from the nose. Coldness of the body presents little difficulty of recognition. The abdomen retains heat the longest and therefore should be felt to establish coldness. A rectal temperature reading is more accurate. Drying of the conjunctivae of the eyes and the formation of a film of inspissated secretion and brownish discoloration of the exposed portion of the eye are confirmatory signs of death.

Estimation of Duration of Death.—No reliable criterion exists for accurate determination of the duration of death. Following death there is, in order of their occurrence, immediate muscular relaxation and pallor, slow development of coldness, lividity and rigor, then muscular relaxation, followed by decomposition or mummification.

Since the *lividity* is the least influenced by the internal condition of the body and medium in which it lies, and is primarily an effect of gravity, it presents a fairly reliable criterion for determining the duration of death during the first hours. Lividity usually appears within two to six hours and progresses to completion within twelve hours. Its appearance is delayed by loss of blood and anemia.

In contrast to lividity, *rigor* is primarily the effect of chemical change and is affected by conditions existing in the body before death and by the temperature and circulation of the medium in which the body lies. The extreme variability of the elapsed time before onset, the time required for completion, and the duration of rigor is so great as to render estimation of time of death most difficult from this finding alone. Rigor is delayed by hemorrhage and is slow to appear in well developed muscles; it is hastened by muscular exertion, exhaustive diseases, and by poor muscular development. Usually, the more rapidly it appears, the more quickly it disappears, and vice-versa.

As a rule, rigor develops first in the muscles of the face and jaws, and then in the order named, in the neck, upper extremities, trunk and lower extremities; it disappears in the same order. It usually develops between two and six hours after death, becomes complete between two and twelve hours, and lasts from one to three days. Low temperatures prolong rigor.

The length of time for the development of *coldness* is also variable. Body heat is lost rapidly during the first hours and slowly after this. Temperature and circulation of the medium in which the body lies and the initial temperature of the body and the extent of clothing and covering greatly affect the rate of cooling.

Decomposition begins after the disappearance of rigor and is similarly influenced regarding interval before appearance, rate of completion, and duration. Since blood dries rapidly, the finding of fluid or clotted blood indicates recent death. Articles about the body, such as dated papers, condition of perishables like food, and other circumstances are valuable in estimating the duration of death and should be considered in conjunction with the condition of the body in making this estimate.

Circumstances of Death.—If a practicing physician is confronted with a suicide or homicide before the police arrive, his examination at the scene of death assumes greater significance than if the police were the first to arrive. In making his examination the physician should avoid any examination that requires *alteration of the original appearance or position.*

For instance, he should not contaminate objects, especially firearms and knives, with his *fingerprints*.

In *bullet wounds*, the location, shape, size, margins, powder marks, and position of the wound in relation to area of the powder marks, have individual significance. The significances of location are chiefly the accessibility of the wound to the hand of the deceased and its relation to vital parts. A bullet striking the skin perpendicularly leaves a wound with round margins; one striking the skin at an angle leaves a wound with margins half round and half oval, the oval half facing the direction of fire. An obliquely striking bullet furrows the skin and leaves a wound with elliptical margins, the long diameter of which lies in the direction of the course of the bullet. The apex of the furrow points in the direction of fire. Wound margins of somersaulting bullets striking flat are elongated—oval at one end and square at the other. Ricocheting bullets produce bizarre shaped wounds. The size of the bullet wound does not correspond, necessarily, to the caliber of the bullet.

The margins of bullet wounds are abraded, contused, and stained by grease and powder and the immediately surrounding tissue is contused. *Black powder* causes an abundance of powder burns and marks on close fire, in contrast to the paucity caused by *smokeless powder*. Perpendicularly fired bullets lie in the central part of the area of powder marks, whereas obliquely fired bullets have an eccentrically placed wound situated away from the direction of fire. The character of the powder marks gives a rough determination of the distance of fire within a distance of several feet. An estimation of this nature, however, requires special knowledge.

Characteristic features of *shot wounds*, owing to the small size and roundness of the shot, are less evident but are generally similar to those of bullet wounds. Shot spreads, for the most part, in direct proportion to the distance of fire. Contact wounds from pistols, rifles or shot guns are large and have torn stellate margins, resulting from the explosive force of the gases introduced in the tissues. The margins of wounds of entrance are inverted, whereas the wounds of exit are everted. Wounds of exit made by lead bullets are usually larger than entrance wounds, and are frequently irregular or torn in several directions. Bullets encased in hard metal have, as a rule, exit

wounds similar to entrance wounds except for the eversion of the margins of exit wounds.

Stab wounds, incised wounds, chopping wounds, contusions, lacerations and *abrasions* have few specific external characteristics other than their individual form. Wounds about the hands and parts of the body relatively inaccessible to the hands, and incised wounds with peculiar patterns or markings like crosses or symbols, suggest homicide. Although a *multiplicity* of wounds is usually associated with homicide, two or more wounds are occasionally found in suicides. The *amount and location of effused blood* and its relation to wounds are significant. A great amount of blood of course suggests exsanguination; blood in the ears suggests fracture of the base of the skull; and, blood stains may reveal inconspicuous wounds. Ecchymosis of the eyelids suggests fracture of anterior part of the base of the skull, in the absence of external evidence of injury about the eyes. Careful examination of the scalp for blood stains may reveal inconspicuous fatal wounds, such as a wound caused by an icepick. The direction of the flow of blood on clothes or body indicates the position of the body during hemorrhage. The presence or absence of perforations of clothes corresponding to wounds in covered parts of body is an important determination. If disturbance of clothing is necessary to make this determination, it is precluded so far as a practicing physician is concerned. Occasionally, sudden collapse or falling in sudden death may result in lacerations which bleed freely, and hence may become confused with those produced by criminal violence.

Asphyxia by hanging, strangulation, or throttling has one common characteristic: the mark made by the constricting object. This is the most reliable sign, externally or internally, in determining such deaths. In *hanging*, the constriction mark is usually found over the entire circumference of neck and includes the mark of the knot. Marks of fingernails and abrasions and contusions by the fingers are characteristic in *throttling*. Constriction marks made by narrow objects like a rope are deep and narrow in contrast to the wide areas of compression and abrasions of skin made by belts, ties, scarfs and similar agents. In *hanging*, the constriction mark of the rope becomes deeper with time, and in a day or two the skin forming

the furrow assumes the consistency and appearance of leather. Antemortem fractures of the hyoid bone and thyroid cartilages are uncommon before death in hanging and in strangulation, as compared to their common occurrence in throttling. In bodies that have remained in a hanging position for a day or two, postmortem fractures occasionally occur as a result of the gradual increase in the constriction. Constriction marks made by rope on the anterior part of neck usually lie between the hyoid bone and laryngeal cartilage, or over the hyoid bone, whereas constriction marks made by belts lie over the laryngeal cartilage. Fractures of the hyoid bone and thyroid cartilage are readily felt. Determination of antemortem or postmortem fractures depends, however, on the absence or presence of hemorrhage at the fracture site. This determination is made only at autopsy and does not directly concern the practicing physician, but hemorrhages in the skin about the constricting mark are equally important. In asphyxiation by *smothering*, no marks of violence are usually found except those made while struggling. Asphyxia by covering is difficult to detect unless coverings and body are intact.

Other important circumstances of death are the *place of death*, general *size of room, state of furniture and bed, dates on newspapers and mail, state of preservation of food*, presence of *smoked cigarettes* and *used matches, bottles labeled as alcoholic beverages, time, weather* and *temperature*. Even more important circumstances are the condition and type of *clothing* on the body and the *relation of the weapons* to the hand and the injuries.

As a rule, no great effort is required to make accurate conclusions, provided proper emphasis is placed on important circumstances. Overemphasizing one circumstance, such as disorder of the furniture in a room, will not occur if directly related circumstances, such as the absence of signs of struggle about the clothes or body, are noted. One of the most valuable single circumstances of suicide by shooting or cutting is of course finding the weapon in the hand or near body of the deceased. The absence of weapons about the body, multiple wounds, wounds about the hands and in regions difficult to reach by the hands of the deceased, and signs of assault, are of course valuable circumstances of homicide. A necessary

circumstance to establish in suicide by hanging, when the body is suspended with the feet off the floor, is the "step-off object." Attempts to disguise homicides as suicides by hanging are often remiss in this respect. No such object is needed when the feet are resting on the floor, since the weight of the partially supported body is sufficient to cause asphyxia by hanging. Owing to the extremely sudden loss of consciousness in hanging there is little opportunity for struggling, even if the suicide attempted to free himself after tightening of the noose. More remote circumstances, such as a previous attempt or threat of suicide, insanity, suicide note, a criminal record of the deceased or a previous threat to murder him, are usually ascertained and investigated by the medical examiner or coroner. Although suicide notes are most valuable in determining suicide, the practicing physician may have access to them only when they are displayed openly. Sealed and addressed suicide notes are to be opened only by the medical examiner or coroner.

Circumstances of death also include the general features of the deceased and specific identifying marks: *Sex, color, apparent age, height and weight, muscular development, complexion, color of hair and eyes* and *bodily deformities* are general means of identification. *Scars, birthmarks, moles,* condition of *teeth, tattoo marks* and *clothing* are more specific means of identification.

Medicolegal Aspects.—As soon as a physician has concluded from his examination that a death is suspiciously like a suicide or homicide, his first duty is to report such a death to the police, medical examiner, coroner or properly designated authority in his community. It is advisable that the physician remain at the scene of death until the lawfully designated investigator arrives, and he should then cooperate with him until released. On the other hand, if a physician is called by the police to pronounce dead a suspected suicide or victim of a homicide, or if the body is brought by the police to the hospital, all available circumstances of death *at that time* should be carefully studied. It is most helpful to the medical examiner or coroner if the physician will keep himself available by phone for several hours after reporting the death. In Maryland, the physician is not specifically required by law to give information obtained by his examination of the circumstances of death

to a medical examiner, but in New York City the medical examiner is permitted by law to subpoena witnesses for the purpose of obtaining information regarding the circumstances of death. Medical examiners and coroners issue death certificates for suicides and homicides. Various penalties exist for falsifying death certificates; in Maryland, a fine of not less than $50 nor more than $200.

Trials of Persons Accused of Homicide.—These usually concern practicing physicians only after the accused has been held for trial in the criminal courts as a result of hearings in lower courts. Suicides, by their nature, rarely result in criminal court procedures. In court trials the practicing physician receives a summons to appear as a witness at a certain time in a designated court, and it behooves him not only to obey the summons, but also to restudy well the information he has about the case in question. A *written record* of the findings of his examination of the circumstances of death should be made as soon as possible after the examination and filed for possible future use.

After the physician is sworn, he is qualified as an ordinary witness or expert witness and establishes his connection with the case. An *ordinary witness* states only facts that he saw or heard during his examination of circumstances of death. An *expert witness* gives opinions deduced from his examination of the circumstances of death or from testimony given by other witnesses. An ordinary witness can be qualified as an expert witness if the court so desires for the purpose of asking an opinion. Physicians as ordinary witnesses are usually paid the customary fee for ordinary witnesses established by the court, while expert witnesses receive fees considerably higher than those of ordinary witnesses, the amount being determined by the court or the attorney for the defense.

Testifying begins with an examination by the state's attorney and ends with the completion of cross examination by the attorney for the defense. *While testifying,* witnesses should face the jury in jury trials, and face the judge in court trials. If the witness does not talk plainly, loudly enough and in language a layman can understand, he will be interrupted by the court, attorneys or jury until he does so. Answers to questions should be brief and free from elaboration. Hearsay is not ac-

cepted as evidence. Incriminating questions need not be answered. If uncertain regarding the correct answer to a question, it is best to say *"I do not know."* Cross examination of ordinary witnesses is relatively brief as compared to that of expert witnesses. In qualifying, an expert witness may be required to relate his educational record, special studies and training, hospital and medical school appointments, contributions to medical literature, and evidences of special knowledge of subjects pertaining to testimony. The court may or may not approve the qualifications. Expert witnesses are expected to give their own opinions, unless various individual authorities are quoted as a uniform opinion and with this opinion the witness agrees. If quotations from a book are presented, the witness should ask permission of the court to read the quoted statements. As a rule it is unwise to quote the opinion of authorities as related above. Voluble witnesses and those with hazy memories as to the facts presented and statements made in the course of the testimony are the delight of a bullying cross examiner. The physician has little or no recourse from such bullying except in the manner in which he gives testimony. The calm, patient, but forcefully speaking witness will avoid most of the machinations of the cross examiner by persistently restating former evidence to questions so posed as to distort the answers previously given. Answers "Yes" and "No" are not mandatory, but an answer to a question is mandatory unless excused by the court. Also, the witness is to address his answers to the jury or judge.

Testimony of *ordinary witnesses* is referable usually to the facts regarding the circumstances of death. Such witnesses may be asked to identify the body, weapons and objects at the scene of death, give the state of muscular development and the size of the body, and describe the wounds and other related matters. A cross examiner may be able to challenge successfully the ability of a witness to identify the deceased. Good muscular development and large size may be stressed through a witness by the attorney for the defense in order to strengthen a plea of self defense, or by the prosecuting attorney to stress willfulness on the part of the accused. *Expert testimony* may range from an opinion of the time of death to a description of a test used for the determination of the percentage of alcohol

in the contents of a bottle. Only an expert in ballistics can qualify in identifying a pistol as the one causing death. An ordinary witness can say, however, that the pistol or knife is similar to the one he saw at the scene of death. Most expert testimony in homicide trials is given by medical examiners, coroner's autopsy physicians, and toxicologists. In Maryland, the medical examiners are qualified by law as expert witnesses.

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CARBON MONOXIDE—A DOMESTIC AND OCCUPATIONAL HAZARD

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Carbon monoxide, a colorless, odorless and tasteless gas, is the most prevalent and most insidious of the toxic gases encountered in modern civilization. Its victims are found in domestic life, in transportation and in many industrial occupations. A product of the incomplete combustion of carbonaceous materials, carbon monoxide occurs in varying concentrations whenever fuels of any type are burned; in the exhaust gases of internal combustion engines; in the firing of explosives, and in certain chemical processes. While natural gas contains no carbon monoxide, manufactured gas, so widely used as fuel in the home and in industry, contains about 20 per cent of this gas as one of its constituents. The inhalation of unburned ("raw") manufactured gas is one of the favorite methods in vogue for committing suicide—death apparently comes painlessly and unconsciousness rather quickly. The frequency of accidental carbon monoxide poisonings is indicated by the large number of such cases reported in the daily newspapers during the fall and winter months.

Historical.—Since man first made use of fire for warmth, and later to cook his food, there have no doubt been poisonings and deaths from exposure to carbon monoxide. Sayers[1] and Davenport, referring to a report of Dr. L. Levin on the history of carbon monoxide poisoning, mention instances described in ancient literature which indicate that this gas was a frequent cause of accidental deaths, was used for suicidal purposes and at times as an instrument of punishment or torture. Exposure of persons to smoke from wood fires or to charcoal vapors in small enclosures apparently was a convenient way of getting rid of undesirables. It is stated that the use of the charcoal

brazier for suicidal purposes became quite popular in France during the nineteenth century.

With the advance in civilization, methods of heating homes improved; new fuels and new equipment were developed for domestic and industrial use; and the consumption of gasoline in internal combustion engines for automobiles, motor boats and aeroplanes reached millions of gallons annually. All of these developments have increased the possibilities of carbon monoxide poisoning. According to reports of the National Safety Council, accidental deaths in the United States from the inhalation of poisonous gas numbered 1704 in 1937 and 1459 in 1938. Unfortunately there are no Federal statistics which separate carbon monoxide poisoning from poisoning by other gases. It is estimated, however, that about 90 per cent of the deaths recorded are due to this gas. In addition to the annual number of deaths from carbon monoxide inhalation, there occur many more nonfatal accidental poisonings.

Some Sources of Carbon Monoxide.—Automobile exhaust gas contains about 7.0 per cent of carbon monoxide. Mine explosion gases, industrial blast furnace gases, and gases manufactured for fuel purposes contain from 8.0 to 30 per cent carbon monoxide. Furnace gases from small house heating systems may contain 1.0 per cent of carbon monoxide, and smoke from burning buildings 0.1 per cent.

Lack of proper maintenance, improper construction or adjustment of appliances and equipment, lack of proper ventilation, defective venting or defective piping, and faulty installation are frequent causes for carbon monoxide poisonings in the home and in industry. Investigation of such cases often reveals that the source of carbon monoxide is from a furnace with a partly blocked chimney or from leaky, deteriorated piping in use for carrying off the incompletely burned gases. Occasionally, flexible gas tubing becomes deteriorated to the extent that unburned gas escapes into the room. Tampering with burners or vents of gas-fired appliances may result in incomplete combustion, attended by excessive amounts of carbon monoxide. Automobile exhaust gas from running motors in closed garages or in repair shops, or the leakage of these gases into the vehicles from defects in the exhaust system result in many cases of carbon monoxide poisoning each year.

Some idea of the sources and frequency of carbon monoxide poisonings in the home and from automobile exhaust gas may be gained from the data given in Table 1, taken from reports of the Ohio State Department of health:[2, 3, 4, 5]

TABLE 1

CARBON MONOXIDE POISONINGS—CASES AND DEATHS

	1937		1936		1935		1934	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
Domestic:								
Bathroom heaters..	5	3	23	4	12	3	3	1
Other room heaters.	32	14	38	20	11	6	23	15
Cooking stoves....	8	2	27	2	21	5	13	4
Water heaters.....	70	11	26	6	18	2	16	3
Other appliances...	13	2	13	1	16	4	16	4
Coal or coke stoves.	24	4	55	5	17	2	16	1
Charcoal burning..	5	4	4	4	1	0	3	0
Furnace..........	..	..	2	0	13	1	2	0
Oil heater.........	..	..	2	2	4	0	1	1
Motor vehicles:								
Garages—residence.	62	45	63	28	33	20	27	15
Public garages.....	19	19	17	1	4	1	6	0
Vehicles en route ..	44	8	43	11	15	1	29	8

Since gases, other fuels and the internal combustion engine are widely used in many industries, the problem of exposure to carbon monoxide is one of first importance to the industrial physician and to industrial management. While the number of fatal poisonings from this gas may be relatively small in industrial occupations, there do occur significant numbers of accidental nonfatal poisonings which result in illness and lost time. Surveys made by state and municipal industrial hygiene units have shown carbon monoxide to head the list of toxic gases encountered in industry. Industries engaged in the production of chemicals, clay and clay products, clothing, iron, steel and other metals, paper and printing, transportation, and domestic and personal service show the percentage of employees with possible exposure to carbon monoxide to range from 1.0 per cent to as high as 38.7 per cent. A number of states now include carbon monoxide poisoning as a compensable occupational disease.

PHYSIOLOGIC EFFECTS OF CARBON MONOXIDE

Carbon monoxide has an affinity for combination with the hemoglobin of the blood approximately 300 times that of oxygen. In an atmosphere containing this gas the oxyhemoglobin of the blood is converted to carbon monoxide hemoglobin, thus excluding the necessary supply of oxygen to the body tissues and ultimately resulting in asphyxiation if the concentration of carbon monoxide in the air breathed and the duration of exposure are sufficient. The reaction between carbon monoxide, oxygen and hemoglobin is a reversible one following the well known "mass law" of chemistry. In the presence of fresh air or pure oxygen the reaction proceeds in the reverse manner, oxyhemoglobin is formed and the carbon monoxide is eliminated. Carbon monoxide is classified as a *chemical asphyxiant*, not as a direct tissue poison.

Apparently no injury is done to the erythrocytes by the combination of carbon monoxide with the hemoglobin. They are, however, deprived of their vital oxygen-carrying function. Subsequent elimination of the carbon monoxide from the blood restores this function to normal capacity. The sequelae of carbon monoxide poisoning are attributed to the extent and duration of the anoxemia caused by the formation of the carbon monoxide hemoglobin. It is for this reason that, in cases of acute poisoning, it is so important to eliminate the combined carbon monoxide from the blood as soon and as rapidly as possible in order to avoid permanent damage to the oxygen-starved tissues.

Rate of Absorption.—The rate at which carbon monoxide hemoglobin is formed in the blood is dependent upon the concentrations of the gas in the air breathed, the duration of exposure, the activity of the individual and to a lesser extent upon age, high temperature, high humidity and individual susceptibility. Activity plays an important role in the rate of absorption because of the attendant increase in respiratory rate and a similar increase in oxygen demand by the body tissues. As the percentage of hemoglobin combined with carbon monoxide increases there is an accompanying increase in the degree of anoxemia which ultimately results in death if the carbon monoxide concentration in the air breathed is sufficiently high. Sayers and Yant[a] have prepared data (Table 2)

which show the predominating symptoms as the percentage of blood saturation with carbon monoxide increases:

TABLE 2

PREDOMINATING SYMPTOMS OF CARBON MONOXIDE POISONING ACCORDING TO THE PERCENTAGE OF BLOOD SATURATION

No symptoms 0–10
Tightness across forehead; possibly slight headache, dilation
   of cutaneous blood vessels 10–20
Headache; throbbing in temples 20–30
Severe headache, weakness, dizziness, dimness of vision,
   nausea and vomiting, collapse 30–40
Same as previous item with more possibility of collapse and
   syncope, increased respiration and pulse 40–50
Syncope, increased respiration and pulse; coma with inter-
   mittent convulsions; Cheyne-Stokes' respiration 50–60
Coma with intermittent convulsions, depressed heart action
   and respiration, possibly death 60–70
Weak pulse and slowed respiration, respiratory failure and
   death 70–80

Elimination of Carbon Monoxide.—*Removal to fresh air* is of first importance in the treatment of carbon monoxide poisoning in order that the replacement by oxygen of combined carbon monoxide in the blood may be accelerated. Under such conditions about 50 per cent of the absorbed carbon monoxide is eliminated from the body during the first hour. However, complete elimination is not obtained for a number of hours, therefore some degree of anoxemia may persist for an appreciable length of time. To speed up the rate of carbon monoxide elimination, *pure oxygen* or a *mixture* of 93 per cent oxygen and 7 per cent carbon dioxide should be administered. The carbon dioxide-oxygen mixture has the advantage over oxygen alone in that it stimulates respiratory activity by increasing the frequency and depth of respiration. For cases of carbon monoxide asphyxiation, equipment for administering this gas mixture is available. It is used in many cities by emergency crews of utility companies, fire departments, police departments and industrial plants.

DANGEROUS CONCENTRATIONS OF CARBON MONOXIDE

It has been stated that the effects of carbon monoxide are dependent chiefly on the concentration of this gas in the air breathed and the duration of exposure. As the result of exten-

sive studies it is now the general consensus that a concentration of 100 parts of carbon monoxide per million parts of air should not be exceeded when the duration of exposure extends over a period of several hours. Concentrations of 400 to 500 parts per million parts of air have no appreciable effect after one hour of breathing, whereas 1500 to 2000 parts per million are dangerous for exposure of one hour and a concentration of 4000 parts per million is fatal in less than an hour.

The *exhaust gas* from the average passenger automobile contains about 7.0 per cent carbon monoxide (70,000 parts per million parts of air) and is sufficient to render the air of a single-car garage deadly in five minutes if the engine is allowed to run when the garage doors are closed. It is not unusual to find concentrations of carbon monoxide exceeding 1500 parts per million parts of air in the products of combustion given off by *defective* industrial and domestic *fuel-burning equipment.* Whether or not acute or mild cases of poisoning occur depends on such factors as ventilation, size of the room, dilution of the escaping gases by the surrounding atmosphere and length of exposure. A defective gas-fired pressing iron may cause severe symptoms in the worker using it while others working at some distance in the same room may complain only of mild headaches or may show no symptoms whatever. In one instance a vent from a kerosene-fired steam boiler in a tailoring shop terminated in an outlet into a bedroom on the upper floor for the purpose of supplying heat. This dangerous installation caused the death of a child sleeping in a crib close by the outlet while the mother in a nearby bed showed only severe poisoning symptoms. In some storage garages the ventilation in use under normal operating conditions may suffice to keep concentrations of carbon monoxide below physiologic limits, while during rush periods symptoms of poisoning are manifested by employees.

Some mention should be made of the various so-called *gas-saving devices*[7] often sold by door-to-door vendors for attachment to domestic gas-fired appliances. A number of these have been found to be extremely dangerous, causing high concentrations of carbon monoxide to be produced by equipment which performs safely without them. In one case a mother and daughter were nonfatally asphyxiated as the result of inserting such a device on the burner of a hot water heater, and many

housewives suffered mild poisoning symptoms after installing these attachments on the burners of their kitchen gas ranges. Gas-fired appliances now on the market are generally safe in operation so long as there is no interference with their normal performance. Attachments should not be added without assurance from a reliable source that they are safe and will not interfere with the proper combustion characteristics of the appliance.

SYMPTOMS AND DIAGNOSIS OF POISONING

Acute Poisoning.—When poisoning by carbon monoxide is rapid there may be an absence of subjective symptoms, the victim being either unconscious or in a state of mental confusion. This acute form of poisoning is manifested by a *progressive paralysis of the central nervous system* and, according to Sayers and Yant,[6] results in an increase in pulse and respiratory rates, fall of blood pressure, loss of muscular control, especially of sphincters, loss of reflexes, coma usually with intermittent convulsions, Cheyne-Stokes' respiration, slowing of pulse, slow and shallow respiration, cessation of respiration, death. If the patient recovers, permanent damage may have been done to the brain and other organs due not to any action of absorbed carbon monoxide but to the insufficient supply of oxygen in the blood. It is the intensity and duration of the period of asphyxiation which are related to such *late symptoms and sequelae* as listed by Mayer[8]:

1. Headaches.
2. General weakness, especially of the muscles.
3. Pains in the limbs, with or without numbness or tingling.
4. Tremor.
5. Palpitation of the heart, with shortness of breath on exertion.
6. Attacks of intense pain or pressure over the region of the heart, resembling angina pectoris.
7. Anemia.
8. Extreme dryness of the throat, sometimes of long duration, causing serious interference with sleep.
9. Various mental or nervous symptoms.

Chronic Poisoning.—While some investigators claim that repeated subacute exposures to carbon monoxide over long periods of time, as may be found in automobile repair shops, around blast furnaces, in clothing manufacturing plants and

in poorly ventilated kitchens, may cause permanent damage and are responsible for such subjective symptoms as *headaches, nausea, irritability, shortness of breath* on exertion and *fatigue,* other authorities deny the occurrence of injury to health from frequent and prolonged inhalations of low concentrations of this gas. Whichever school of thought is correct, there is ample evidence that long periods of exposure to low concentrations of carbon monoxide are responsible for distressing symptoms which interfere with the general well-being of the individual. As an example, in a tailoring shop a presser using a gas-fired iron complained of distressing gastro-intestinal disturbances, particularly nausea and loss of appetite. Tests showed the iron to be defective and the worker exposed to significant concentrations of carbon monoxide. When the appliance was replaced by one of satisfactory performance the symptoms disappeared.

The difficulty in establishing a diagnosis of chronic exposure to carbon monoxide is evident from the nature of the symptoms which are common to a great variety of disorders. Workers in a garage may complain of headaches, gastro-intestinal disturbances, dizziness or fatigue which may or may not be associated with carbon monoxide in the working environment. It is of utmost importance in order to arrive at a correct diagnosis in such cases that exposure to carbon monoxide be demonstrated by *air analyses* and by *examination of the blood* of the employee for its carbon monoxide content. The blood sample, of course, must be taken during or immediately after the exposure because of the instability of the carbon monoxide hemoglobin on removal of the person from the atmosphere containing carbon monoxide.

While the symptoms of chronic exposure to carbon monoxide are common to many health disorders, a careful occupational history combined with a study of the individual's environment will often indicate whether this gas is involved. Industrial chronic exposures to carbon monoxide are relatively easy to detect and their control and prevention are comparatively simple. Present-day standards for gas-fired appliances together with approved methods for their venting, and the installation of adequate ventilation in garages and in other industrial operations where carbon monoxide may be a hazard

have gone a long way toward the control of exposures to this gas both in the home and in the workroom.

Methods for Detecting Carbon Monoxide.—In view of the difficulty in establishing a diagnosis of carbon monoxide poisoning by observation of subjective symptoms alone, a knowledge of, and the ability to apply, the simpler methods for the detection and determination of this gas are of importance. A brief description of several of these methods and their application follows:

Pyrotannic Acid Method.[9, 10]—This method is very useful for determining the carbon monoxide saturation of the blood of a person who has been exposed to the gas, and can likewise be used for estimating low concentrations in the air. The necessary equipment is small and compact and may be readily carried in a coat pocket. The method is based on the formation of a light-carmine (red) suspension when a mixture of pyrogallic and tannic acids is added to a water solution of blood that contains carbon monoxide combined with the hemoglobin. Normal blood under similar treatment causes a light brownish-gray suspension. The intensity of the red color produced is proportional to the percentage saturation of the blood with carbon monoxide. This percentage is determined by comparing the color in the sample treated as described with a set of permanent color standards that range, in steps of 10, from 0 to 100 per cent saturation.

For determining air concentrations of carbon monoxide, a sample of the atmosphere is collected in a bottle of known capacity, preferably ranging from 100 to 250 cc. A solution of 0.1 cc. of blood (human or animal) diluted to 2 cc. with water is introduced into the bottle containing the sample, and the bottle is then rotated, in a horizontal position, for fifteen to twenty minutes. The blood solution, which now contains the carbon monoxide hemoglobin formed by exposure to the air sample, is transferred to a small test tube and the mixture of pyrogallic and tannic acids is added. The percentage of saturation of the blood with carbon monoxide is determined by comparison with the color standards previously mentioned, and from this the concentration of carbon monoxide in the air sample is calculated. The equipment is inexpensive and offers wide application in the investigation of exposures to carbon

monoxide and in establishing this gas as a cause of subjective symptoms and of deaths in fatal poisonings.

Ampule-type Carbon Monoxide Detector.[11, 12]—Such a detector is useful for testing air in garages, tunnels, in sewer manholes, around blast furnaces and gas plants, and in residences. A solution of palladium chloride in a water-acetone mixture comprises the testing agent. This is contained in small, cotton-covered, thin walled ampules, hermetically sealed. The testing procedure consists of crushing the ampule, wetting the cotton covering with the palladium chloride solution, then exposing it for ten minutes in the atmosphere suspected of containing carbon monoxide. Depending on the concentrations of carbon monoxide present, the changes in the color of the ampule range from a yellowish black to black. The concentration of carbon monoxide in the air being tested is estimated by comparing the color of the ampule after exposure with a color chart which is supplied with each package of ampules.

This method gives semiquantitative estimations of carbon monoxide concentrations ranging from 200 to 1000 parts per million parts of air. The sensitivity of the ampule detector decreases with low temperatures but satisfactory results may be obtained if the exposure is extended to from twenty to thirty minutes at temperatures ranging between 50° and 32° F. The test is unreliable at 0° F. The color change is based on the reduction of palladium chloride to a black precipitate of metallic palladium by carbon monoxide. Gasoline vapor, ethylene, hydrogen and hydrogen sulfide produce similar discolorations of the ampule, but only when present in poisonous or explosive concentrations.

Portable Carbon Monoxide Indicator.[12]—This is mentioned here because of its wide applicability, its sensitivity, and ability to read the concentrations of carbon monoxide in the atmosphere on a meter when a sample of air is drawn directly into the apparatus. Concentrations of carbon monoxide ranging from less than 100 parts per million to 1500 parts per million can be determined. This equipment is useful in making studies of carbon monoxide exposures in airplanes, in closed automobiles, in garages and automobile repair shops, in motor boats, in residences, and in fact in almost any place where the gas is suspected. It has advantages over the methods

previously described in that the concentrations of carbon monoxide present are known within a few minutes after operating the equipment and variations in amounts of the gas at different locations are readily determined.

FIRST AID TREATMENT

Victims of carbon monoxide poisoning should be *removed to the fresh air* as soon as possible.

In those cases where breathing is difficult or has stopped *artificial respiration* by the Schaefer method should be given until normal breathing is restored or until the heart has stopped.

Administration of a mixture containing 93 per cent *oxygen* and 7 per cent *carbon dioxide,* or of pure oxygen by means of an inhalator should be begun as soon as possible. Too much stress cannot be placed on the need for promptness in this procedure. Delay may result in severe after-effects.

The patient should be *kept warm* by the use of blankets, hot water bottles or other heated devices, precautions being taken however that hot objects are wrapped so as not to produce burns.

Complete rest, lying down, should be practiced to avoid any strain on the heart. While exercise hastens the elimination of carbon monoxide from the body it is dangerous and the victim may lose consciousness. In some instances deaths have resulted from such practice.

It is of utmost importance to remember that the object of treatment is to remove the carbon monoxide from the blood in the shortest possible time. The longer the tissues are deprived of their normal supply of oxygen the greater are the possibilities of permanent after-effects.

PREVENTION OF CARBON MONOXIDE POISONING IN THE HOME

Inspection of Heating Plants.—There exists a possibility of exposure to carbon monoxide whenever any fuel is burned, since this gas is almost always present in varying concentrations in the products of combustion. So long as these gaseous waste products are conveyed safely to the outdoor air, or the equipment operates in such a manner that the carbon

monoxide concentration in the room does not exceed 100 parts per million parts of air, there is no danger. Quite frequently, during those periods of the year when artificial heat is needed in the home and little outdoor ventilation is provided, accidental carbon monoxide poisonings occur from some defect in fuel-burning equipment. Many of such cases could be prevented by making periodic inspections of furnaces to see that flues and vent pipes are kept clean and are in a good state of repair, and that the openings in chimneys are not obstructed by accumulations of soot or other objects which might prevent the free escape of smoke and gases. Coal gas frequently contains dangerous concentrations of carbon monoxide. Fortunately its presence can usually be detected by the odor of other substances in this gaseous mixture.

Use of Approved Gas-burning Equipment.—Gas-burning appliances are widely used in the home. Cooking ranges, room heaters, water heaters, furnaces, clothes dryers and other equipment operated by gas as a fuel have made domestic life more comfortable. Faulty design, improper installation or adjustment, deterioration or clogging of vital parts and carelessness or ignorance regarding operation are the more important reasons for gas appliances being responsible for carbon monoxide poisonings. To combat these dangers so far as the fixtures and their installation are concerned many cities have passed ordinances for the protection of the public. Baltimore, Maryland,[18] adopted such an ordinance in July, 1925, after an extensive study was made by the Health Department of the increasing number of fatalities attributed to the use of defective gas appliances. This ordinance requires that all appliances, tubing, appurtenances or devices to be used with manufactured gas must be approved by and registered with the Commissioner of Health before they are eligible for display, sale or installation; dealers in appliances are required to be licensed; and gas fitters must pass an examination for registration as evidence of their knowledge of the operation and installation of gas-burning equipment.

The gas industry has taken the initiative in endeavoring to provide the public with equipment that operates safely. In 1925, the American Gas Association established *testing laboratories* in Cleveland, Ohio, where manufacturers can submit

their appliances for tests pertaining to safety and efficiency. Rigid specifications must be met in order to obtain approval and the privilege of attaching to the equipment the approval seal of the American Gas Association. Many cities now require such approval for gas appliances sold and installed within their jurisdictions. By providing these safeguards around the types of gas-burning equipment sold to the public, a noteworthy advance has been made in the control of carbon monoxide hazards in the home.

Educational methods are a necessity for acquainting the public with possible dangers from the use of resold deteriorated appliances and from tampering with the vital parts of gas-burning equipment. In one instance an entire family was made ill over a period of several weeks from the use of a second-hand gas range. A section of the oven lining had deteriorated to the extent that it was dislodged and partly smothered the flame of the oven burner causing the production of significant amounts of carbon monoxide. Readjustments of burner orifices by un-skilled persons supplying excessive or overloading amounts of unburned gas to burners are quite frequently a cause for acci-dental poisonings. Warnings of the dangers and sources of carbon monoxide in the home and information on precautionary measures for guarding against poisoning by this gas should be broadcast at every possible opportunity.

PREVENTION OF CARBON MONOXIDE POISONING IN INDUSTRY

Many industrial occupations entail possibilities of exposure to carbon monoxide. While acute poisonings of industrial origin may be comparatively rare, the subacute or repeated and prolonged exposures to low concentrations are of frequent oc-currence in such places as automobile storage and repair shops, garment and hat manufacturing plants and foundries. The industrial physician should have a thorough knowledge of all operations in the plant and should pay particular attention to those occupations which entail a possible carbon monoxide hazard. It has previously been stated that the carbon mon-oxide concentration in the air should not exceed *100 parts per million parts of air* for exposures of eight hours' or more dura-tion. A safe policy is to endeavor not to exceed this concen-tration at any time.

Studies made by the Baltimore City Health Department of the working environments in the *clothing manufacturing* industry and in *tailoring shops* in Baltimore, Maryland, revealed exposures to carbon monoxide in connection with the use of gas-fired pressing irons, flexible gas tubing and gas and kerosene fired steam boilers. Actual field tests made in a number of workrooms showed concentrations above the physiologic limit in several instances. Improper design, faulty installation and neglect of proper maintenance were the chief causes of existent hazards.

In large workrooms where carbon monoxide is being introduced by faulty equipment the severity of the poisoning may vary from mild to acute forms, depending upon such factors as proximity to the source of the gas, activity of the workers and ventilation. In one such workroom, employees about 25 feet from an improperly operated gas-fired steam boiler showed acute poisoning symptoms while a group of pressers nearby complained only of mild headaches. Air currents from an open window were found to be responsible for the variations in the concentrations of carbon monoxide in the workroom air.

Proper design, proper installation, proper maintenance and periodic inspection of fuel-burning industrial equipment are fundamental in the prevention of carbon monoxide poisoning in industrial plants.

In public garages and repair shops automobile exhaust gas is the source of carbon monoxide. Cases of poisoning in varying degrees of severity are most likely to occur during the winter months when outside ventilation is restricted. The presence of other compounds in the exhaust gas, such as benzol, may cause poisoning symptoms in addition to those of carbon monoxide alone. Adequate ventilation, properly applied, is essential for keeping exposures within safe limits. When it is necessary to run the engine for testing purposes, and the vehicle is in a stationary position, the best control measure is to attach flexible tubing to the exhaust pipe of the automobile and to a duct which is designed to exhaust the gases to the outside air. For vehicles in motion a detachable vertical exhaust pipe should be attached to the exhaust pipe of the car so that the gases from the car are discharged above the breathing level of the workers. Artificial exhaust ventilation applied at

the ceiling of the room then serves to remove the accumulated gases to the outside air by suction.

COMMENT AND SUMMARY

Carbon monoxide, a colorless, odorless and tasteless gas, is frequently encountered under a diversity of circumstances in domestic and industrial life. Burning fuels of any type, operation of internal combustion engines and a number of industrial processes entail the possibility of exposure to this gas. Many deaths and nonfatal poisonings are caused each year by exposures to dangerous concentrations of carbon monoxide. This gas having much greater affinity for combination with the hemoglobin of the blood than oxygen when inhaled, causes an anoxemia the degree of which depends chiefly on the concentration of carbon monoxide in the air breathed and the period of the exposure. The sequelae of carbon monoxide poisoning are attributable to the extent and duration of the anoxemia.

Where exposures continue over a period of several hours the carbon monoxide concentration in the air should not exceed 100 parts per million parts of air. Acute poisoning is manifested by a progressive paralysis of the central nervous system ultimately resulting in unconsciousness and death. Subacute poisonings are characterized by such subjective symptoms as headaches, nausea and vomiting, weakness, dimness of vision and dizziness. For establishing a diagnosis of carbon monoxide poisoning the blood of the patient should be examined for its carbon monoxide content and its source should be determined.

Victims of carbon monoxide poisoning should be removed to the fresh air as soon as possible. If breathing is difficult or has stopped artificial respiration should be given and either a mixture of 93 per cent oxygen and 7 per cent carbon dioxide or pure oxygen should be administered by means of an inhalator. The patient should be kept warm and quiet until fully recovered.

The use of properly constructed, properly installed and properly maintained fuel-burning equipment and the effective venting of such equipment as may be required are the chief preventive measures against carbon poisonings in the home

and in industry. Exposure to automobile exhaust gas, which is rich in carbon monoxide content, should be avoided by never running engines in closed private garages, by making certain there is no leakage of this gas into the automobile as the result of defects, and by providing sufficient natural and artificial ventilation in public garages and automobile repair shops. Continued educational methods designed to acquaint the public with the sources, symptoms and dangers of carbon monoxide poisoning both in the home and in industry should help to reduce the large number of fatal and nonfatal poisonings caused by this gas each year.

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THE USES AND ABUSES OF THE SULFONAMIDE DRUGS*

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Less than six years have elapsed since Domagk[1] published his original observations on the striking effects of the brick-red dye Prontosil upon experimental streptococcal infections and thereby introduced a new era in the chemotherapy of bacterial infections. This important communication marks the starting-point of a stupendous amount of experimental and clinical investigation of the sulfonamide group of drugs. As a result, within a very few years many new compounds have been synthesized, tested out in the laboratory, and rendered available to the clinician for the treatment of various types of infections. Among the important compounds that have been developed are *sulfanilamide, neoprontosil, sulfapyridine, sulfathiazole,* and most recently *sulfanilyl-guanidine.*

Perhaps the chief problem which confronts the clinician to-day is the selection of the *proper drug* for the treatment of a *particular infection.* The purpose of this paper is therefore twofold: (1) to discuss the indications for the use of the different sulfonamide drugs, and (2) to point out certain abuses of these drugs and contraindications to their administration.

SULFANILAMIDE

On the basis of our present knowledge, sulfanilamide is still the drug of choice for the treatment of infections due to the hemolytic streptococcus, meningococcus, Ducrey bacillus and

Welch bacillus. The therapeutic value of this drug is also well established in certain infections of the urinary tract, and in trachoma and lymphogranuloma inguinale.

Dosage and Administration.—In order to administer sulfanilamide in the most effective manner the clinician must be familiar with certain facts regarding the *absorption* and *excretion* of the drug. On the basis of experimental and clinical observations, certain standards of sulfanilamide therapy have been formulated by Long, Marshall and their associates.[2] By checking the concentration of sulfanilamide in the blood of human beings after the administration of a single dose of the drug by mouth, they found that the peak blood level of sulfanilamide was reached in four hours. This observation forms the basis for their contention that sulfanilamide should be given *in divided doses* administered at four-hour intervals, *both day and night*, in order to maintain the concentration of the drug in the blood at the desired level. In Tables 1 and 2 are listed the doses which Long and Bliss[3] believe to be therapeutically effective in severe and mild infections caused by the microorganisms enumerated above. These are not hard and fast rules, but will serve as a general guide to dosage, subject to modification when the individual case demands it.

In severe infections it is important to attain an effective blood level as soon as possible. Therefore a large initial dose of sulfanilamide is recommended in order that the desired level of 10 mg. per cent may be attained as quickly as possible. This level should be maintained or increased by doses of the drug given at four-hour intervals both day and night. The maintenance dose should be continued until a marked clinical improvement in the condition of the patient is noted. Then sulfanilamide should be decreased slowly day by day, but not discontinued entirely until the patient is ready to be up and about. If this routine is followed, recurrences of infection will be rare. It is important to remember that sulfanilamide should be discontinued only under the most exceptional circumstances in the face of a severe infection.

In the milder tissue infections susceptible to sulfanilamide therapy, blood levels of the drug of from 5 to 10 mg. per cent are generally adequate to bring the infection under control. Here again it is important to maintain an even concentration of

the drug in the tissues, and because of this a four-hour dosage schedule is best if circumstances permit its use.

If the patient cannot swallow tablets, if vomiting be present, or if there is any reason to believe that the absorption of

TABLE 1

THE AMOUNTS OF SULFANILAMIDE NECESSARY TO ESTABLISH THERAPEUTICALLY EFFECTIVE BLOOD LEVELS (10 TO 15 MG. PER CENT) QUICKLY IN PATIENTS ILL WITH SEVERE HEMOLYTIC STREPTOCOCCAL, MENINGOCOCCAL, GONOCOCCAL, PNEUMOCOCCAL OR WELCH BACILLARY INFECTIONS*

Weight of Patient		Initial Dose per Os		Maintenance Dose per Os Q 4 Hours (Day and Night)		Total Dose First 24 Hours		Total Daily Dose Bicarbonate of Soda	
Kilos	Pounds	Grams	Grains	Grams	Grains	Grams per Kilo	Grains per Pound	Grams	Grains
70	150	4.8	80	1.2	20	0.15	1.2	3.6	60
60	125	4.2	70	0.9	15	0.15	1.2	3.0	50
45	100	3.6	60	0.9	15	0.18	1.3	3.0	50
35	75	3.6	60	0.9	15	0.23	1.8	3.0	50
23	50	3.0	50	0.6	10	0.26	2.0	1.8	30
11	25	1.8	30	0.3	5	0.3	2.2	0.9	15

* After Long, P. H., and Bliss, E. A.

TABLE 2

THE AMOUNTS OF SULFANILAMIDE NECESSARY TO ESTABLISH THERAPEUTICALLY EFFECTIVE BLOOD LEVELS (5 TO 10 MG. PER CENT) IN PATIENTS ILL WITH MILD OR MODERATELY SEVERE TISSUE INFECTIONS IN WHICH SULFANILAMIDE THERAPY IS INDICATED*

Weight of Patient		Calculated Daily Dose				Dose Per Os Q 4 Hours (Day and Night)		Total Daily Dose Bicarbonate of Soda	
Kilos	Pounds	Grams	Grams per Kilo	Grains	Grains per Pound	Grams	Grains	Grams	Grains
70	150	5.4	.07	90	0.6	0.9	15	3.6	60
60	125	5.4	.09	90	0.7	0.9	15	3.6	60
45	100	5.4	.12	90	0.9	0.9	15	3.6	60
35	75	4.2	.12	70	0.9	1 of 1.2*	1 of 20	2.4	40
						5 of 0.6	5 of 10		
23	50	3.6	.16	60	1.1	0.6	10	1.8	30
11	25	1.8	.16	30	1.2	0.3	5	1.2	21

*After Long, P. H., and Bliss, E. A.

sulfanilamide from the gastro-intestinal tract might be faulty, the drug may be given *by hypodermoclysis*. In the preparation of sulfanilamide for parenteral use, a one-sixth molar solution of sodium racemic lactate (18.67 gm. of sodium racemic lactate

per liter) is the recommended solvent, although physiologic saline may be used. It has been found expedient to use a 1 per cent solution of sulfanilamide, and this may be sterilized by boiling for three minutes at 15 pounds pressure. The calculated daily dose is the same as that for the first day of the oral dosage. The initial hypodermoclysis should contain at least half of the calculated first day dose of sulfanilamide. Subsequent hypodermoclyses should be given at eight-hour intervals and should contain one third of the calculated first day dose of sulfanilamide. The parenteral use of sulfanilamide is, however, a less satisfactory method of administering the drug, and in every instance, as soon as the patient is able to take sulfanilamide by mouth at regular intervals the parenteral use of the drug should be discontinued.

It will be noted in Tables 1 and 2 that the amount of sulfanilamide per pound or kilogram of body weight required to establish adequate levels of the drug in the blood of children is considerably greater than that needed in adults. This variation is due to the fact that the fluid intake per kilogram of body weight is normally greater in children than in adults, and when fever is present this difference is even more marked. Sulfanilamide is excreted almost entirely in the urine. Thus, the greater the volume of urine, the greater the excretion of sulfanilamide. This raises the question as to *how much fluid should be given* to a patient who is receiving sulfanilamide. Experience definitely shows that if large amounts of fluid are given, then it is difficult to obtain and maintain effective levels of sulfanilamide because the drug is rapidly excreted. Because of this, fluids should not be forced too strenuously upon a patient who is receiving sulfanilamide.

Since sulfanilamide easily passes over into the spinal fluid in about the same concentration as exists in the blood, *intrathecal* therapy with the drug is not necessarily indicated in meningeal infections. Sulfanilamide can be found in about the same concentration as it exists in the blood in transudates and exudates into all of the body cavities. It also penetrates into the pus in closed abscesses and is present in purulent discharges. In the treatment of *purulent infections*, recurrences are frequent if sulfanilamide is discontinued before a complete clinical cure has been effected. Thus, in draining abscesses,

in streptococcal osteomyelitis and in middle ear or mastoid infections the drug should be continued for at least *ten days* after the patient is completely well. Where there are localized collections of pus in streptococcal infections, as for example, in one of the paranasal sinuses, *surgical drainage* of the pocket of pus should be instituted early as an adjunct to sulfanilamide therapy. A possible exception to the rule arises in the case of mastoiditis where it appears safe to try sulfanilamide alone for two or three days in the hope that the necessity for surgical drainage will be averted.

There does not appear to be any contraindication to the administration of *other drugs* during the course of sulfanilamide therapy. Aspirin, the barbiturates, digitalis, arsphenamine, ferrous sulfate and many other drugs may be used in conjunction with sulfanilamide if they are indicated. Bicarbonate of soda should always be administered when sulfanilamide is being given in order to prevent a clinical acidosis from developing. The *antidote* for sulfanilamide is water, of which large quantities should be administered to the patient if it is desired to free him of sulfanilamide rapidly.

Hemolytic Streptococcal Infections.—Sulfanilamide should be employed in the treatment of all infections caused by the beta hemolytic streptococcus. At the present writing, there is no evidence that any of the other sulfonamide drugs is superior to sulfanilamide in the treatment of hemolytic streptococcal infections.

Streptococcal Sore Throat, Scarlet Fever, and Complications.—A sufficiently large number of cases of streptococcal sore throat and scarlet fever have been treated with sulfanilamide to justify the following conclusions: In the treated cases the febrile course is shorter, the soreness of the throat disappears more rapidly, and the complications are fewer than in the untreated cases. Certainly the swollen tender cervical nodes tend to melt away as if by magic under sulfanilamide therapy. All observers are agreed that sulfanilamide has no effect upon the toxemic features of scarlet fever.

It is important to emphasize here that, although clinical recovery from streptococcal sore throat may occur very rapidly, the throat culture may remain positive for streptococci for weeks afterwards. In other words, it appears that sulfanilamide

enables the body to get rid of the streptococci that have invaded the tissues but that the drug is incapable of ridding the surface of the body of bacteria. The mucosa of the pharynx, the crypts of the tonsils, and the cavities of the paranasal sinuses would all fall into the category of areas outside the body or on the surface of the body where the bacteria may take refuge from the damaging effects of sulfanilamide.

Longcope[4] has stressed this principle of the control of the invasive properties of hemolytic streptococci by sulfanilamide. He has further noted that sulfanilamide appears to shorten the course of glomerular nephritis secondary to hemolytic streptococcal infections. Of special interest is the fact that the exacerbation of the nephritis which so frequently follows tonsillectomy in such patients may be prevented by the administration of sulfanilamide during the pre-tonsillectomy and post-tonsillectomy periods.

The dosage schedule outlined in Table 2 should prove adequate for the treatment of the majority of hemolytic streptococcal infections of the upper air passages.

Erysipelas, Streptococcal Cellulitis, Lymphangitis and Lymphadenitis.—Erysipelas is the condition par excellence in which hemolytic streptococci invade the tissues of the host. It is probably for this reason that erysipelas responds more dramatically than any other type of streptococcal infection to sulfanilamide therapy.

Streptococcal cellulitis, lymphangitis and lymphadenitis also subside rapidly under sulfanilamide therapy. Because these tissue infections are often fulminating in character, it is advisable to follow the dosage schedule outlined in Table 1.

Hemolytic Streptococcal Meningitis.—Before the introduction of sulfanilamide therapy, meningitis due to the hemolytic streptococci constituted one of the most serious and rapidly fatal of all infections, with a case fatality rate of between 95 and 100 per cent. The case fatality rate has dropped to about 25 per cent since the introduction of sulfanilamide. Obviously, the dosage schedule in Table 1 is indicated in such severe infections. As previously noted, there is no advantage in administering sulfanilamide intrathecally; however, it may be necessary to give the drug by hypodermoclysis because of coma or vomiting.

Puerperal Sepsis Due to Hemolytic Streptococci.—It was the English report[5] of the striking beneficial effects of prontosil and later sulfanilamide in hemolytic streptococcal puerperal sepsis that really stimulated the introduction of chemotherapy into this country. Subsequent experience with sulfanilamide in such infections has borne out that of Colebrook and Kenny. Once more the dosage schedule in Table 1 should be employed.

Hemolytic Streptococcal Empyema.—Hemolytic streptococcal empyema presents a difficult therapeutic problem, especially in adults. Sulfanilamide should be administered in dosage sufficient to maintain the blood level of the drug between 10 and 15 mg. per cent and pleural fluid should be removed by repeated thoracentesis. If in spite of such measures the temperature remains elevated, the cultures of the pleural fluid remain positive, and the exudate becomes thicker and more purulent, more radical methods of drainage must be instituted in order to prevent pocketing of pus. There appears to be no advantage to injecting a solution of sulfanilamide directly into the pleural cavity since the drug passes rapidly from the blood into the pleural exudate where it attains a concentration almost as high as that present in the blood.

Meningococcal Infections.—Waghelstein[6] has treated a series of 106 cases of meningococcal meningitis with sulfanilamide, with nineteen deaths. There appeared to be no advantage in combining serum therapy with sulfanilamide or in the intrathecal administration of the drug. The sulfanilamide seemed more effective when administered orally or by nasal catheter than by the subcutaneous route. Waghelstein's conclusions that sulfanilamide therapy has reduced the number of deaths, not only in the adequately treated cases, but also in those fulminating cases which usually succumb within twenty-four hours, are in accord with the observations of other workers in the field. The dosage in Table 1 should be followed.

Chancroid (Soft Chancre).—In 1938 several reports of the rapid cure of chancroid with sulfanilamide appeared in the British and American literature. Kornblith and his associates[7] advise using 4.8 gm. (80 grains) of sulfanilamide per day in four divided doses for the first five days and then 2.4 gm. (40 grains) a day for the next ten days. The patients were likewise instructed not to drink more than 1 quart of water

a day. The only other treatment advised was the use of soap and water to cleanse the local lesion twice daily. The average duration of treatment was fifteen days and an average amount of 41.4 gm. (690 grains) of sulfanilamide was given. The average time for healing was about two weeks. There appeared to be no contraindication to the simultaneous administration of antisyphilitic treatment to those patients afflicted with both syphilis and chancroid.

Favorable results have been claimed for *local treatment* of the chancroidal ulcer by packing it with sulfanilamide. Although such a measure may prove to be a useful adjunct to oral administration of the drug, it should never be used alone since the bacteria deeper in the tissues could not be reached by this means and buboes would not be affected.

Welch Bacillary Infections.—*Gas gangrene,* due to infection of injured tissue with the anaerobic Clostridium welchii, has long been regarded by surgeons as one of the most dreaded complications of severe traumatic lesions like compound fractures. Treatment of such injuries in the past has consisted in immediate débridement of dead or crushed tissue and prophylactic injection of antitoxin. In spite of these precautions, gas gangrene was prone to develop in a fairly large proportion of such cases, and, once it was apparent that gas gangrene had set in, immediate amputation of the affected limb was usually performed as a life-saving measure. Indeed, it has always been considered a conservative procedure to amputate an extremity in which gas gangrene had made its appearance.

Today, since Bohlman's[8] initial report in 1937 of the successful treatment with sulfanilamide of three patients suffering from gas gangrene and the repeated confirmation of his observations by other orthopedic surgeons, a heavy responsibility rests upon the surgeon who advises the amputation of an extremity because of a gas bacillus infection. Exceptions might be made when the diagnosis of the infection has been missed and the process is far advanced, or when there is no possibility of obtaining a functional restoration of the injured limb.

In an adult patient weighing 150 pounds, 90 to 120 grains (5.4 to 7.2 gm.) of sulfanilamide a day should be administered in six doses at four-hour intervals along with 10 grains (0.6

gm.) of sodium bicarbonate at each dose. The drug should be continued in the original dosage for four days after the temperature has been normal and all signs of gas gangrene have disappeared. The dose should then be reduced slowly over a period of one week; if signs of relapse develop, intensive therapy should be resumed. Surgical débridement of the affected area should be carried out as an important adjunct to sulfanilamide therapy in all cases.

Long and Bliss go one step further and urge that all patients who have had crushing injuries be given *prophylactic doses* of sulfanilamide (60 to 90 grains a day) as soon as they are seen. It seems highly probable that many of the serious complications of wartime injuries may be prevented by the prompt prophylactic administration of sulfanilamide in addition to tetanus antitoxin.

Lockwood[9] only recently has presented an excellent discussion of the use of sulfanilamide in surgical conditions. He has been disappointed in the effects of the drug in experimental gas bacillus infections.

Urinary Tract Infections.—The successful treatment of urinary tract infections with sulfanilamide depends to a large degree upon obtaining and maintaining a satisfactory concentration of sulfanilamide (nonacetylated) in the urine. Satisfactory urine levels of sulfanilamide cannot be obtained in patients who have moderate or marked degree of impairment of renal function. Patients with impaired renal function must always be observed carefully while receiving sulfanilamide, lest the drug accumulate in their tissues. Long and Bliss have noted that the following urinary concentrations of free sulfanilamide are generally effective in the control of urinary tract infections: for Staphylococcus albus infections, 50 to 100 mg. per cent; Staphylococcus aureus, 100 to 150 mg. per cent; Bacillus coli, 200 to 250 mg. per cent; B. lactis aerogenes, 250 to 300 mg. per cent; group B beta hemolytic streptococci, 250 to 300 mg. per cent, and B. proteus infections, 300 to 400 mg. per cent. Enterococcal infections of the urinary tract are unaffected by sulfanilamide therapy.

With a given dose of sulfanilamide and a fairly constant urinary output, it takes from three to four days to reach a balance between the intake of the drug by mouth and the out-

put in the urine. It is therefore advisable to limit fluids in adult patients until the daily urinary output is around 1000 cc. Then, depending upon the type of infecting organism, enough sulfanilamide is given in four equal divided doses (with half or equal doses of sodium bicarbonate to prevent acidosis) so that by the third or fourth day of treatment the required amount of unaltered sulfanilamide is present in the urine. As a rule, one half of the sulfanilamide in the urine is present in the unaltered (free) form, while the other half exists in the conjugated state (acetyl-sulfanilamide) which is inactive against bacteria. The desired concentration of free sulfanilamide should be maintained in the urine for seven days. Clinical observations tend to show that therapeutic effects are not obtained until adequate concentrations of free sulfanilamide in the urine are reached. Sterilization of the urine should be the only criterion of cure.

Alyea and Roberts[10] have recently claimed good results with smaller doses of sulfanilamide and forcing fluids; such a regimen might be recommended for patients who must remain ambulatory during treatment.

Trachoma and Lymphogranuloma Inguinale.—Although we have had practically no personal experience in the treatment of trachoma or lymphogranuloma inguinale, the reports in the literature of the striking effects of sulfanilamide on these infections are so convincing that they must be accepted as conclusive. Sulfanilamide must be administered over relatively long periods in treating both of these infections.

Toxic Effects of Sulfanilamide.—It is well known that sulfanilamide may give rise to a number of toxic manifestations such as anorexia, nausea, dizziness, mental confusion, cyanosis, acidosis, drug fever, dermatitis, hepatitis, hemolytic anemia and granulocytopenia. The first six of the toxic manifestations enumerated are not serious and they will not be discussed in detail here. *Acidosis* may be prevented by administering sodium bicarbonate along with sulfanilamide as outlined in the dosage tables. If *drug fever, dermatitis, hepatitis, acute hemolytic anemia* or *granulocytopenia* develop, the drug should as a rule (always in the case of granulocytopenia) be discontinued and fluids forced to rid the body of the drug as rapidly as possible. Transfusions may be necessary in cases of severe hemo-

lytic anemia. It is important to follow the temperature, the hemoglobin level and the leukocyte count at regular and frequent intervals in order that the more serious toxic effects may be recognized in their incipiency and the drug discontinued before further damage has been done.

SULFAPYRIDINE AND SULFATHIAZOLE

Since the indications for the use of sulfapyridine and sulfathiazole are almost identical, these two drugs will be discussed together. They have been found to be distinctly superior to sulfanilamide in the treatment of pneumococcal, staphylococcal and gonococcal infections and appear to be just as effective as sulfanilamide in certain other infections.

. **Properties and Administration.**—As in the case of sulfanilamide, it is important for the clinician to be familiar with certain special properties of sulfapyridine and sulfathiazole if he is to employ these compounds successfully.

Unfortunately, sulfapyridine possesses several characteristics which make it more difficult to administer than sulfanilamide. Sulfapyridine is *much less soluble* than sulfanilamide, and it is absorbed with less regularity. It is therefore impossible to predict what blood level may be expected in a given individual from a certain dose of the drug. Even in the absence of vomiting the amount of the drug absorbed may vary from 30 per cent of the administered dose to 80 per cent in two different patients. Furthermore, *conjugation* of sulfapyridine in the body to the inactive acetylated form is highly variable in different subjects, so that it is important to determine the level of free sulfapyridine in the blood at frequent intervals in order to be certain that an effective blood level is being maintained.

Blood levels of 4 to 6 mg. per cent of free sulfapyridine are considered satisfactory for the control of mild to moderately severe pneumococcal infections, whereas a blood level between 7 and 12 mg. per cent is desirable in more severe infections. It has been found expedient to grind up the tablets of sulfapyridine and to suspend the powder in water, milk or other fluid for oral administration, as this measure may facilitate the passage of the drug through the pylorus and hasten its absorption from the intestine. Applesauce and jam have

proved useful vehicles for the administration of the powder to children.

Because of the low solubility of sulfapyridine the *parenteral* administration of the drug itself is impractical. Marshall[11] has prepared a soluble sodium salt of sulfapyridine which may be administered intravenously in 5 per cent solution. Within a very short time after a solution of the sodium salt is injected, the sodium ion is split off and the substance circulating in the blood is sulfapyridine. In preparing sodium sulfapyridine for intravenous use, the required amount of the drug is weighed out and dissolved in enough sterile distilled water to make a 5 per cent solution. Sodium sulfapyridine is unstable to heat and hence such solutions cannot be sterilized. However, as such a solution is highly alkaline, it is in itself somewhat bactericidal. The solution should be injected slowly (not more than 5 cc. per minute); since it is very alkaline, care must be taken to avoid extravasation into the tissues lest sloughing occur.

Sulfathiazole is more rapidly absorbed and more rapidly excreted than sulfapyridine. Because of the rapid excretion it may be difficult to maintain the concentration of free sulfathiazole in the blood at the desired level, even though conjugation to the inactive acetylated form is usually less marked than in the case of sulfapyridine.

During the administration of sulfapyridine or sulfathiazole, *fluids should be forced* sufficiently to maintain the twenty-four-hour output of urine between 1000 cc. and 1500 cc. in an adult patient. This measure is important in order to prevent if possible the precipitation in the kidneys of crystals of acetyl-sulfapyridine or acetyl-sulfathiazole, which is more likely to occur if the urine becomes highly concentrated. Since clinical acidosis has not been noted during the administration of either of these sulfonamide compounds, there is no need to give sodium bicarbonate along with them as in the case of sulfanilamide. Other drugs may be administered in conjunction with sulfapyridine or sulfathiazole if they are indicated.

Pneumococcal Pneumonia.—On the basis of the experimental investigations published to date, there seems to be little choice between sulfapyridine and sulfathiazole for the treatment of pneumococcal infections. In clinical lobar pneumonia

the temperature may fall to normal a little more rapidly in patients treated with sulfapyridine, but this questionable advantage is more than equalized by the relatively low incidence of vomiting in sulfathiazole-treated cases. Hence, sulfathiazole may be regarded as the drug of choice for treating pneumococcal pneumonia.

Sulfapyridine or sulfathiazole should be administered to all patients with acute lobar pneumonia, bronchopneumonia or postoperative pneumonia as soon as the diagnosis is established clinically. The drug should also be given in cases of severe purulent bronchitis where a pneumococcus is suspected as the etiologic agent. Before administration of the drug is started, sputum should be obtained for culture and pneumococcus typing and a blood culture should be taken if laboratory facilities are available.

ADMINISTRATION AND DOSAGE.—A large initial dose of 4 gm. (or 60 grains) of the drug is advisable for adult patients in order to establish an effective level of the drug in the blood as soon as possible. Following this large initial dose, a maintenance dose of 1.0 gm. (15 grains) should be given every four hours night and day until the temperature has remained normal for seventy-two hours. The drug may then be discontinued entirely. If such a program is followed, relapses may occur in about 5 percent of patients but the dangers of drug toxicity are materially lessened. *Relapses,* when they occur, may usually be controlled by reinstituting chemotherapy promptly. There is distinct danger of relapse if the drug is discontinued too early in the course of convalescence. In the treatment of *children* Long[12] recommends 1 grain per pound as an initial dose and ¼ grain per pound every six hours as a maintenance dose. Barnett and his associates[13] suggest a dosage schedule for infants and children which proved highly satisfactory in their hands.

The above dosage schedule should prove adequate for the treatment of patients moderately ill with pneumococcal pneumonia. *In severely ill patients,* especially those with bacteremia, it is desirable to obtain a blood level of 7 to 12 mg. per cent of the free compound as soon as possible and to maintain this level until evidence of improvement is apparent. It is in such cases that the intravenous administration of sodium

sulfapyridine has proved most valuable. The sodium salt should be given intravenously in dosage of 0.06 gm. per kilogram of body weight at the initial injection. This dose may be repeated in four hours if advisable; it is then usually possible to place the patient upon an oral maintenance dose of 1.0 gm. of sulfapyridine or sulfathiazole every four hours as described above. In Table 3 the dosage schedule for pneumococcal pneumonia is outlined in condensed form.

TABLE 3

DOSAGE SCHEDULE FOR TREATMENT OF PNEUMOCOCCAL PNEUMONIA WITH SULFAPYRIDINE OR SULFATHIAZOLE

Moderately Ill Patients	Initial Dose	Maintenance Dose
(a) Adults.......... ...	4.0 gm. (60 grains)	1.0 gm. (15 grains) every 4 hours night and day until temperature has remained normal for 72 hours. Then discontinue drug.
(b) Children under 30 pounds in weight..............	0.12 gm. per kilogram (or 1 grain per pound)	0.03 gm. per kilogram (or ½ grain per pound) every 6 hours until temperature has been normal for 72 hours.
Severely ill patients...	0.06 gm. per kilogram (or ½ grain per pound) of sodium salt in 5% solution intravenously; may repeat injection in 4 hours.	Drug given orally as above.

SIDE-EFFECTS OF SULFAPYRIDINE AND SULFATHIAZOLE AND THEIR MANAGEMENT.—Sulfapyridine and sulfathiazole may give rise to all of the toxic effects which have been noted during the administration of sulfanilamide, with the possible exception of acidosis. Headache, dizziness, nausea and vomiting, cyanosis, simple fever, dermatitis, mild and acute hemolytic anemias, leukopenia and granulocytopenia following sulfapyridine have all been reported.

Severe *nausea and vomiting* are unfortunately very common during the course of sulfapyridine administration and may present a serious problem, requiring intravenous fluids to prevent dehydration. It has been shown that this phenomenon is largely due to an effect of the drug upon the central nervous system and not to local gastric irritation. If vomiting is marked, it is advisable to omit one dose of the drug before resuming regular treatment. In cases of persistent intractable vomiting the oral administration of sulfapyridine should be

discontinued. One of the following three substitutive measures may be adopted: (1) intravenous administration of sodium sulfapyridine, (2) intravenous administration of type-specific antipneumococcal serum, or (3) oral administration of sulfathiazole.

The incidence of severe nausea and vomiting in patients treated with sulfathiazole is far lower than that in sulfapyridine-treated cases. On the other hand, *cutaneous complications* have been more common in patients treated with sulfathiazole. Among the latter are conjunctivitis and a peculiar eruption resembling erythema nodosum.

Severe *renal damage* (characterized by hematuria, oliguria and nitrogen retention) from the formation of crystals of acetyl-sulfapyridine or acetyl-sulfathiazole constitutes another serious complication that may be encountered. If gross hematuria develops or the nonprotein nitrogen of the blood rises, the drug should be discontinued at once and fluids forced to 5000 cc. or more.

In order that the various toxic manifestations of sulfapyridine or sulfathiazole may be detected early, the patient should be carefully supervised while the drug is being administered. *Proper supervision* should consist of careful clinical observations of the patient, regular temperature recordings, a hemoglobin determination and leukocyte count at least every other day, a daily urine examination for gross hematuria and repeated determinations of the nonprotein nitrogen of the blood, especially if the urine volume diminishes. If any of the more serious toxic manifestations develop, the drug should be discontinued immediately and fluids forced.

SERUM VS. SULFAPYRIDINE AND SULFATHIAZOLE.—In view of the striking therapeutic effects of sulfapyridine and sulfathiazole in lobar pneumonia the question has very naturally been raised: Is the use of antipneumococcal serum to be abandoned altogether? This question may be answered emphatically in the negative. In the first place, neither drug can be regarded in any sense as a sure cure of pneumonia. Secondly, the mechanism of recovery in cases of pneumonia treated with either compound is undoubtedly similar in many respects to spontaneous recovery or recovery in serum-treated cases, in that it depends at least in part upon specific antibody

formation. This hypothesis appears fairly certain even though an excess of antibody may not be demonstrable in the serum at the time of crisis. The failure to demonstrate excessive antibody in the serum at this time does not mean that antibody may not be present in the lungs. Thirdly, MacLeod[14] has shown that certain strains of pneumococci are "sulfapyridine-fast"; these strains are not influenced by sulfapyridine either in cultures or in experimental infections.

Present evidence points to a *combination* of chemotherapy with the administration of type-specific antipneumococcal serum as the ideal method, at least from the theoretical point of view, of treating all cases of pneumococcal lobar pneumonia. Since serum is so expensive and frequently is difficult to obtain, it seems justifiable to treat milder cases of pneumonia with the drug alone. However, in all seriously ill patients, especially those with bacteremia, and in all cases of severe pneumonia complicating pregnancy, chemotherapy should be supplemented from the start with serotherapy if type-specific serum is available. Furthermore, any patient who fails to show improvement within forty-eight hours after the institution of chemotherapy should receive serum.

TREATMENT OF COMPLICATIONS.—*Empyema.*—If empyema is present at the time of admission or develops during treatment, chemotherapy should be continued until the temperature has been normal for at least one full week and repeated cultures of the pleural fluid have remained sterile. It is usually necessary to establish some form of drainage of the empyema cavity in addition to administering the drug. Drainage should be established as soon as a positive culture has been obtained from the pleural cavity in order to prevent pocketing of the pus if possible.

Sterile Pleural Effusion.—Sterile pleural effusions are not uncommon complications of lobar pneumonia. Such effusions do not require drainage unless they become sufficiently large to embarrass respiration, in which case the fluid may be removed by thoracentesis. If the temperature has returned to normal after the development of a sterile effusion and the lungs are clear, chemotherapy may be safely discontinued. .

Pneumococcal Endocarditis.—This is one of the most serious complications of lobar pneumonia, with a fatality rate of

nearly 100 percent. If such a complication is suspected from the course of the temperature, the appearance of cardiac murmurs, and persistent bacteremia, intensive drug therapy should be continued in full dosage. Even so, the prognosis of this complication will undoubtedly remain very grave.

Pneumococcal Meningitis.—The prognosis of meningitis due to the pneumococcus has always been considered to be practically hopeless, particularly when it develops as a complication of lobar pneumonia. The rare recoveries that had been reported prior to the introduction of chemotherapy with the sulfonamide drugs usually occurred in cases of meningitis complicating infections of the middle ear or paranasal sinuses, and even in such cases the case fatality rate was well above 90 percent. Recently Hodes[15] published a series of seventeen cases of pneumococcal meningitis treated with sulfapyridine with eight recoveries, giving the amazingly low case fatality rate of 53 per cent for this small but significant series. The intravenous administration of sodium sulfapyridine solution appears to be the logical method of early treatment in such cases. This solution is much too alkaline to be given intrathecally. However, there appears to be no necessity for employing intrathecal chemotherapy, since the drug rapidly passes over from the blood stream into the spinal fluid where it will attain a level between 60 and 75 per cent of the concentration in the blood.

RESULTS OF CHEMOTHERAPY IN PNEUMOCOCCAL LOBAR PNEUMONIA.—Very recently the results obtained in the treatment of 3005 typed cases of pneumococcic pneumonia with sulfapyridine have been summarized in the most complete survey[16] to appear to date. The case fatality rate for the entire group was only 6.0 per cent; in the 240 bacteremic cases the case fatility rate was 23.3 per cent; in the 2765 nonbacteremic cases 4.5 per cent. Such figures signify a reduction of the former case fatality rate for lobar pneumonia by at least two thirds. Experience to date with sulfathiazole suggests that an equally low case fatality rate may be expected with this compound.

Staphylococcal Infections.—Experimental observations have shown that sulfapyridine and sulfathiazole are far more effective against the staphylococcus than is sulfanilamide, and there is considerable evidence that sulfathiazole is superior to

sulfapyridine. Therefore, in all severe staphylococcal infections, including septicemia, pneumonia and empyema, sulfathiazole should be the drug of choice.

In severe staphylococcal infections, an initial dose of 4.0 gm. of sulfathiazole should be followed by a maintenance dose of 1.0 gm. every four hours night and day. If the clinical response to this dose is not satisfactory, the maintenance dose should be increased to 1.5 to 2.0 gm. every four hours until improvement is definite; it may then be reduced to 1.0 gm. every four hours once more and the latter dose should be continued until the temperature has remained normal for five days in staphylococcal pneumonia. In staphylococcal osteomyelitis and septicemia, sulfathiazole should be continued until the temperature has been normal for two weeks. All purulent foci should be drained where possible as an adjunct to chemotherapy.

Urinary Tract Infections.—Sulfathiazole has been found to be very effective in combating certain infections of the urinary tract, especially where a staphylococcus, Bacillus coli, B. pyocyaneus, or B. proteus is the causative agent. Further clinical studies must be carried out before the apparent therapeutic superiority of sulfathiazole over sulfanilamide in such infections is firmly established.

Gonococcal Infections.—Although there have been a great many reports on the efficacy of sulfanilamide against gonorrhea, it is now generally conceded that both sulfapyridine and sulfathiazole are superior. For either drug the recommended schedule of dosage is as follows: 3.0 gm. the first day, 2.0 gm. a day for the next nine days. If at the end of ten days there is still evidence of active infection, a shift should be made from sulfapyridine to sulfathiazole or vice-versa.

Every patient should be impressed with the importance of avoiding any form of overindulgence, taking no alcohol, and employing measures to avoid infecting his sexual partner for at least three months after sulfonamide therapy. The minimal criteria of cure should be repeatedly negative clinical and laboratory examinations (cultures as well as smears) for three to six months after the cessation of therapy. If these requirements are routinely adhered to, then few symptomless carriers will be turned loose to infect the public.

The septic complications of gonorrhea such as gonococcal peritonitis, arthritis and endocarditis will usually require more vigorous treatment with sulfapyridine or sulfathiazole over longer periods of time.

OTHER SULFONAMIDE DRUGS

Neoprontosil.—Neoprontosil breaks down to release sulfanilamide in the body. As far as can be determined, sulfanilamide is the principal (if not the only) bacteriostatic compound released by this dye in the body. Therefore it is difficult to see how the administration of neoprontosil would offer any advantage over the administration of the simpler compound, sulfanilamide, and as a matter of fact there is no convincing clinical evidence to support the claims that have been made for the greater therapeutic efficacy and the lesser toxicity of neoprontosil.

If the parenteral use of sulfanilamide is considered imperative and facilities are not available for the administration of a large hypodermoclysis, then it may be found convenient to employ the more soluble neoprontosil which is available commercially in sterile ampules. In an adult weighing 150 pounds 10 cc. of a 5 per cent solution of neoprontosil (or 20 cc. of a 2.5 per cent solution) should be given subcutaneously every four hours.

Sulfanilyl-guanidine.—Marshall[17] has recently reported upon the pharmacology of sulfanilyl-guanidine, a sulfanilamide derivative with certain peculiar properties which suggest that it may prove valuable in treating *infections of the intestinal tract.* This compound is much more soluble in water than sulfapyridine, yet it is very poorly absorbed from the intestine. Hence it is possible to obtain a high concentration of the drug throughout the entire length of the intestinal tract. Sulfanilyl-guanidine has been shown to exert a marked bacteriostatic action on the normal intestinal flora as well as intestinal pathogens. The results of the clinical trial of this drug in such infections as bacillary dysentery, cholera and ulcerative colitis will be awaited with much interest.

INFECTIONS IN WHICH SULFONAMIDE DRUGS ARE OF QUESTIONABLE VALUE

In contrast to the infections which have already been discussed in which one or another of the sulfonamide drugs has been shown to be of definite therapeutic value, there are a number of other infectious diseases in which the efficacy of those drugs must still be regarded as questionable. Certainly in such infections the response to chemotherapy is far less dramatic and less regular than in the infections previously enumerated.

Among the infections in which the therapeutic response must be regarded as questionable are many *bacillary infections* such as undulant fever, tularemia, infections with Friedländer bacillus or H. influenzae; also subacute bacterial endocarditis due to Streptococcus viridans and fungus infections such as actinomycosis. Highly suggestive reports have appeared in the literature, claiming good results from chemotherapy in all of these infections, yet in other clinics treatment with the sulfonamide drugs has proved disappointing in these same infections.

Since untreated *subacute bacterial endocarditis* is almost uniformly fatal and a few recoveries have been reported following treatment with sulfanilamide, further careful use of this drug or one of its derivatives seems justified, provided it does not add to the discomfort of the patient. The drug must be continued for weeks or even months in such cases. In a number of instances the temperature has fallen to normal and blood cultures have become temporarily sterile, but the great majority of patients treated have not been materially benefited. Actual recoveries attributed to chemotherapy have not exceeded 5 per cent.

In *undulant fever* sulfanilamide will, as a rule, bring the temperature down to normal and alleviate symptoms; however, relapses are common after the drug has been discontinued. Sulfanilamide should be administered for at least six weeks to patients with undulant fever; large doses should be given until the temperature has been reduced to normal and the dose should then be gradually tapered off during the ensuing weeks.

Further cautious and controlled trial of the sulfonamide drugs is justified in bacterial infections where experimental and

clinical evidence suggests that these compounds may be of therapeutic value.

PROPHYLACTIC USE OF THE SULFONAMIDE DRUGS

Because of the many toxic effects of the sulfonamide drugs, the prophylactic use of these compounds should be limited to those special situations where there is good reason to believe that such prophylaxis will be attended by success. Of the few such situations which exist the following are the most important:

Inactive Rheumatic Heart Disease.—Thomas and France[18] in Baltimore and Coburn[19] in New York have presented convincing evidence that the administration of sulfanilamide during the school year to children with inactive rheumatic heart disease may serve to prevent them from contracting hemolytic streptococcal infections of the upper respiratory tract with the exacerbations of rheumatic fever which are so prone to follow. Since these children appeared to tolerate the drug well, further trial of such a procedure is certainly justifiable.

It is common knowledge that subacute bacterial endocarditis not infrequently develops *following dental extractions* in patients with chronic valvular heart disease. Therefore the prophylactic administration of sulfanilamide in moderate doses for several days before and after dental extractions in such patients may help prevent the settling of micro-organisms on the damaged valves during the transient bacteremic period which so frequently follows the extraction of abscessed teeth.

Scarlet Fever and Other Hemolytic Streptococcal Epidemics.—When an epidemic of scarlet fever or other hemolytic streptococcal infection breaks out in an institution or a household, it appears logical to administer sulfanilamide prophylactically to all exposed individuals for one week in the hope of curtailing the spread of the epidemic.

Crushing Injuries.—As previously pointed out, the immediate administration of sulfanilamide to victims of serious crushing injuries such as compound fractures may prevent the development of dangerous secondary infection, particularly with the hemolytic streptococcus or the Welch bacillus.

Bowel Resection.—Surgeons[20] have found that the administration of one of the sulfonamide drugs (especially sul-

fathiazole) before and after resection of a portion of the bowel helps to cut down the danger of postoperative peritonitis.

ABUSES OF THE SULFONAMIDE DRUGS

The exhibition of any of the sulfonamide drugs in infections in which there is no experimental or clinical evidence that such therapy will prove of value must be regarded as an abuse of the drug in question.   Furthermore, there is no justification for the administration of these drugs in any noninfectious disorder. The drugs of this group are far too toxic to permit of their use indiscriminately without a specific indication.

Probably the most widespread abuse of the sulfonamide drugs to date has occurred in victims of the *common cold* or *influenza*.   Both of these infections are caused by filtrable viruses and there.is absolutely no experimental or clinical evidence that any of the sulfonamide compounds exerts a beneficial effect upon such infections.   Only if a secondary infection with the hemolytic streptococcus or pneumococcus develops is there a justification for the institution of sulfonamide chemotherapy.

What has been said about the common cold and influenza applies to virus infections in general, with the exception of lymphogranuloma inguinale.   In other words, there is no excuse for the administration of the sulfonamide drugs in uncomplicated *measles, mumps, chickenpox, poliomyelitis, encephalitis* or other virus infections.

On the basis of published observations, sulfanilamide, far from exerting a beneficial effect in *active rheumatic fever,* may actually prove harmful in this relatively common infection. The differential diagnosis between acute rheumatic fever and acute gonococcal polyarthritis frequently proves difficult.   In such a situation, every effort should be made to distinguish between these two conditions and to rule out rheumatic fever before sulfonamide therapy is resorted to.   The prophylactic use of sulfanilamide in inactive rheumatic heart disease has been discussed above.

Although there is evidence that sulfanilamide may exert a favorable influence upon experimental tuberculosis in animals, the use of the drug in *clinical tuberculosis* has met with uniformly disappointing results.   Hence, sulfanilamide cannot be

recommended in uncomplicated tuberculosis in human beings. Sulfanilamide has had no effect whatsoever on either experimental or clinical *syphilis;* it appears unlikely that any of the other sulfonamide drugs will affect infections with the Treponema pallidum.

To date none of the sulfonamide compounds has proved effective against *enteric fever* of the typhoid-paratyphoid type or against the various forms of *dysentery.* There is reason to hope, however, that the new compound sulfanilyl-guanidine may be of value in treating these infections of the intestinal tract.

No conclusive evidence has been produced as yet to show that any of the sulfonamide drugs will protect an individual against acquiring *gonorrhea.* Since exposure to an infection of this type must be regarded as chronic in the majority of individuals concerned, the prophylactic use of a sulfonamide compound can scarcely be recommended.

TOXIC EFFECTS OF THE SULFONAMIDE DRUGS

Unfortunately, the sulfonamide drugs give rise to a variety of toxic manifestations, the majority of which have already been mentioned briefly. Long and his associates[21] have very recently summarized these toxic effects in a comprehensive article based on extensive clinical experience. Table 4, taken from this excellent paper, epitomizes the data which they have collected and furnishes abundant evidence why: (1) the sulfonamide drugs should not be employed indiscriminately; (2) these drugs should be administered only under the direct supervision of a physician. Before administering one of the sulfonamide drugs to a patient, it is important to inquire whether he has previously been treated with such a drug and if so whether he suffered from any serious toxic effect. If he has, he should be given a small test dose (0.3 gm. or 5 grains) of the drug before full dosage is instituted.

CONCLUSIONS

There are widespread indications for the use of the sulfonamide drugs in a variety of bacterial infections. It is important to select the most effective drug for the treatment of a given infection.

TABLE 4

MANIFESTATIONS OF DRUG TOXICITY NOTED IN HOSPITALIZED ADULTS; 1000 TREATED WITH SULFANILAMIDE, 297 TREATED WITH SULFAPYRIDINE AND 271 TREATED WITH SULFATHIAZOLE‡

Reaction	Sulfanilamide	Sulfapyridine	Sulfathiazole
Nausea, vomiting	Fairly common	Frequent	Uncommon
Dizziness	Common	Common	Uncommon
Psychoses*	0.6%, occur early	0.3%, occur early	Not reported as yet
Neuritis†	Very rare	Not reported	Not reported
Cyanosis	Very common, early and late	Faint, common, early and late	Uncommon
Acidosis*	1.9%, occurs at any time, rare if soda is used	Not reported	Not reported
Fever*	10%, generally 5th to 9th day, may occur 1st to 30th day	4%, generally 5th to 9th day, may occur 1st to 30th day	10%, generally 5th to 9th day
Rash*	1.9%, may take any form, generally 5th to 9th day, may occur 1st to 30th day	2%, may take any form, 5th to 9th day, may occur 1st to 30th day	5%, nodular type common, may take any form, 5th to 9th day
Hepatitis†	0.6%, early or late	Not seen, but reported	Not reported
Leukopenia with granulocytopenia†	0.3%, early or late	0.6%, early or late	1.6%, early or late
Acute agranulocytosis†	0.1%, occurs 14th to 40th day, common 17th to 25th day	0.3%, occurs 14th to 40th day, common 17th to 25th day	Not reported
Mild hemolytic anemia	3%, early and late	Rare	Not reported
Acute hemolytic anemia†	1.8%, occurs 1st to 5th day	0.6%, occurs 1st to 5th day	Not reported
Hematuria*	Not reported	8%, generally early	2.5%, generally early
Anuria with azotemia†	Not reported	0.3%, generally early	0.7%, generally 1st 10 days
Hyperleukocytosis*	Generally in presence of acute hemolytic anemia	Generally 1st 10 days	Not reported
Injection of sclera and conjunctivas*	Not reported	Generally in presence of acute hemolytic anemia	Not reported
Purpura haemorrhagica†	Not seen, but reported	Not reported	4%, may occur with rash and fever, 5th to 9th day
Ocular and auditory disturbances*	Rare	Rare	Not reported
Jaundice†	With acute hemolytic anemia or hepatitis	With acute hemolytic anemia or hepatitis	Not reported
Painful joints*	Reported	Not reported	Reported with rash, etc.
Stomatitis*	Rare	Not reported	Not reported
Gastrointestinal tract disturbances*	Bleeding rare, diarrhea uncommon	Rare	Not reported

* Best to stop drug and force fluids.
† Imperative to stop drug and force fluids.
‡ After Long, P. H., Haviland, J. W., Edwards, L. B. and Bliss, E. A.[31]

These drugs should not be employed indiscriminately for the treatment of infections upon which they exert no therapeutic effect or in which their use may prove actually harmful or dangerous.

Because of the possible toxic manifestations of the sulfonamide compounds, patients receiving any of these drugs should, whenever possible, be hospitalized in order to insure careful clinical and laboratory control.

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PHYSICAL THERAPY: ITS PLACE IN GENERAL PRACTICE

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We live, fortunately, in an age of great therapeutic interest. We are becoming accustomed to the rapid substitution of better, newer methods in the place of others formerly considered satisfactory. We have come a long way from the age of therapeutic nihilism that reached its peak, no doubt, in the Viennese school where it was said that no patient could be more highly honored than to have the diagnosis made by Skoda and the autopsy by Rokitansky. Things are quite different now and we do not neglect therapy but we run some risk, through lack of interest, of failing to avail ourselves of some less spectacular but nevertheless quite useful methods. I am taking the liberty of presenting to you a sort of orientation exercise in regard to one branch of therapeutics.

PAST AND PRESENT ATTITUDES

Physical therapy is a type of useful treatment applicable to many different ailments. In some form or other it has persisted from ancient times in the healing art, now highly respected, now frowned upon, and so may be said to have had a long and checkered career. It has had its bright moments. Although not particularly in the good graces of the medical profession in the early nineteen hundreds it yet served in part, at least, as the vehicle for two Nobel laureates. Finsen received the prize in 1903 following his demonstration of the curative action of ultraviolet light on lupus vulgaris, and Windaus, on another occasion, for his chemical studies on the sterols including the demonstration of the relationship of ultraviolet radiation and the formation of vitamin D.

By reason of the neglect by the medical profession, physical therapy has been highly exploited by extramural cults and no doubt very often with highly beneficial results both to the healer and to the patient. There are, however, obvious reasons why helpful methods of therapy should not be neglected by physicians and surgeons or resigned to the use of irregular practitioners. As you well know, a sustained campaign has been under way for some time by organized medicine to bring physical therapy back into the medical fold and, with ups and downs and backing and filling, the campaign is bearing fruit. Orthopedists and general surgeons seem to feel the need of physical therapy more than do the internists and this its proponents would like very much to change and to see a real and sturdy interest on the internists' part instead of the present scarcely desultory one.

I cannot claim that I as an internist made the discovery of physical therapy through any special prescience of my own. It was really brought rather forcibly to my attention by the request that I organize and direct a hospital department of it. Although my duties are chiefly administrative they have led to considerable interest in the work and persuaded me that internists looking further into this subject may find much of interest to themselves and of helpfulness to their patients.

I think of a good hospital department as consisting chiefly of a staff of well trained and skillful technicians under competent medical supervision for whom the hospital has furnished the apparatus and materials that will best facilitate their work. Proper records should be kept and opportunities for clinical research should be utilized. The situation is quite different with the general practitioner who is treating patients in their homes. Here there need not be skilled technicians nor expensive equipment but the physician, if he is interested and informed, can teach his patients and responsible members of the family many efficient methods of applying heat and utilizing the simpler types of massage and of muscular exercise, the real fundamentals of physical therapy. Unfortunately, however, the only stimulus and instruction that he now receives come from the salesmen of appliances whose interest is chiefly in the sale of the equipment and whose teaching naturally relates to the use of that probably expensive machine. In only

a few states have the State Medical Societies sponsored addresses and demonstrations on physical therapy before the county and other local societies.

THE NEGLECT OF PHYSICAL THERAPY BY THE MEDICAL SCHOOLS

Recent graduates of medical schools have had little better instruction than the older men. Here let me quote from a paper by Alan Gregg entitled "Addenda to the Agenda for the Decade 1940–1950." This paper was presented by the Director of the Medical Sciences, Rockefeller Foundation, before the Thirty-sixth Annual Congress on Medical Education and Licensure, in February of last year. Its purpose, the author stated, was to offer an inventory of special opportunities for development in medicine in the decade now before us. His paragraphs devoted to physical therapy have been much quoted but are well worth repeating. I quote:

"A curious phenomenon in American medicine is the deft elimination of much reference to physical therapy. Almost as those who keep their children ignorant of the facts of life, we appear to protect the American medical student from the knowledge of physical therapy, mindful, I suppose, of the abuses imputed to osteopaths and chiropractors. Of course there is much the same result: thus protected, our graduates angrily complain of competition from those whose knowledge it is taboo to acquire. Cornford comments on propaganda as the art of lying in such a way that you very nearly deceive your friends without quite deceiving your enemies. That reminds me of the way physical therapy is propagated in this country. But the effects of heat on capillary circulation, lymph flow, and inflammation, and the indications and effects of massage and passive movements, or of irradiation, can certainly be learned and taught as physiology without any trace of charlatanry. The current unabashed ignorance of physical therapy in this country leaves it as an excellent opportunity for development in American medicine."

"Of course, a much more inclusive and significant development in which physical therapy would be but a small part would be the recognition of biophysics on a basis similar to that of biochemistry. True, it would belong close to physi-

ology and at first would derive from physiology as did bio-chemistry. Indeed at the present time the application of methods of electrical stimulation, records, and measurements dominates the research interest of so many American physiologists that biophysics removed from physiology would leave something of a vacuole. As in most of the preceding suggestions, I argue here for the explicit acknowledgment of insidious reality; physics is applied to medicine, physics is required for entrance to medical school, and yet deliberate recognition or energetic development of the potentialities of biophysics is left as the responsibility of no one in particular, leading a grant-in-aid existence until its value as a companion piece of biochemistry is finally admitted."

Very few medical schools at present offer satisfactory courses in physical therapy to their undergraduates—satisfactory in the sense of affording them adequate opportunities to learn the indications, the possibilities, and the limitations of this form of therapy. The crowded condition of the medical curriculum is a formidable difficulty in the introduction of any new course. Many schools, however, are now paying some attention to this problem. Eight medical schools now offer postgraduate courses where physicians who are particularly interested in its application may undertake intensive study of physical therapy for several weeks or months.

THE PERSONNEL FOR PHYSICAL THERAPY

What of the personnel now devoting themselves to this work?

The American Medical Association has officially exerted a most beneficent influence during the last decade, largely through its Council on Physical Therapy composed of physicians, surgeons, physiologists, pathologists, and only two specialists in physical therapy. The work of the Council has been sane and conservative and of great value in the acceptance or refusal of approval of apparatus, the stimulation of research, and interest in education. The Handbook of Physical Therapy published by the Council and frequently revised is a useful small volume for the physician's desk as well as a textbook for the medical student.

The Paucity of Specialists.—No great number of physicians have as yet specialized in this field. Coulter in an article on medical preparedness states that in 1938 a group of physicians interested in physical therapy organized a special society for physicians specializing in physical therapy, the Society of Physical Therapy Physicians. The requirements for membership in this society are that members shall have been engaged in the practice of physical therapy for at least five years and shall have given evidence of satisfactory scientific attainments. In 1940 this society had a membership of thirty-eight and a waiting list of ten. It is obvious from these figures that there are not many men whom they would consider suitable to head the physical therapy departments of large hospitals in either military or civil practice.

Trained Technicians.—There is a shortage also in well trained technicians but it seems unwise to lower the standards that exist at present. My observation of them during the past few years persuades me that they form an admirable group, young, enthusiastic, devoted and ethical. They are recruited from the ranks of registered graduate nurses or from college graduates who have majored in physical education. With this educational background, they are given an extra academic year in physical therapy during which they add to their knowledge of anatomy and physiology and acquire training in the necessary technical procedures. There are now sixteen schools approved by the Council on Medical Education and Hospitals. On graduation from an approved school, the candidate may take the examinations given by the American Registry for Physical Therapy Technicians, sponsored by the American Congress of Physical Therapy, and also those of the American Physiotherapy Association. Taking into account the overlapping in these two organizations, Coulter estimates that there are about 1275 trained physical therapy technicians in the United States. Among their ethical requirements is the provision that they will practice only under the supervision and direction of physicians or surgeons. These technicians are now employed in hospitals, in schools for crippled children, in the State Program Services for crippled children, and in physicians' private offices.

THE OUTLOOK

Progress is being made in building up this branch of therapeutics by the profession. It is no longer necessary to see one's patients slipping away to irregular practitioners.

A glance at the *legislative* situation quickly reveals that physical therapy is, in most states, practiced by the medical profession, by osteopaths, by chiropractors, by beauty parlors, by health institutes, by reducing agencies, and still others. Naturally, it is the desire of interested physicians that physical therapy should be a definite part of medical practice. Every effort should be made to save it from lay practice. With this in mind it is thought inadvisable to license even well trained technicians. There are problems in education. Situations will arise in the legislative field that will require careful watching and consideration. You are men of influence in your communities, academic and political. There may well occur opportunities in which your interest and influence would be of great value.

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CONVULSIONS IN INFANCY AND CHILDHOOD
With Special Reference to Treatment

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A THOROUGH discussion of all the conditions which precipitate convulsions in infancy and childhood is obviously impossible within the limits of a short monograph. The author has responded to a request for a résumé on the subject of the treatment of convulsions with this article, emphasizing therapy, but in which diagnosis is also stressed, for the obvious reason that the *prime factor in the proper treatment of convulsions is the ability to discover their cause.* The treatment of the seizure itself is of minor importance.

The specific syndromes which include convulsions are described, the causative mechanisms discussed, and therapy outlined; the first two as succinctly as is possible without omitting salient facts. Obviously not too great detail can be entered into, and for this reason key references to the literature have been inserted at various points. A complete review of the literature has not been attempted.

IMMEDIATE TREATMENT OF THE CONVULSION

The traditional advice of the experienced practitioner to "dress carefully and smoke a large cigar" before rushing to see a child with convulsions has some merit. Certainly most seizures are short and self-limited and death practically never occurs during them. However, details of the attack may in certain instances prove exceedingly valuable indicators of the correct diagnosis, and the family is in no condition to make or remember accurate observations. Therefore, this excellent advice must be disregarded.

Telephonic instructions should be simple. The child is to be sponged with, or immersed in a tub, of tepid or *cool water*. Hot mustard baths should not be employed, since further elevation of an already high fever may be dangerous. A *lukewarm saline enema* may be given.

The salient features of the convulsion having been carefully noted, the administration of sedatives is in order. Intramuscular *phenobarbital* (grain ¼ to 1), *chloral hydrate* (grains 3 to 10) by rectum, or small doses of *morphine* (grain ¹⁄₁₀₀ to ¹⁄₁₂) often suffice. If these have no effect within 10 to 15 minutes *ether* or *chloroform* should be administered. It is not safe, from the point of view of possible cerebral damage by the convulsion itself, to allow the seizure to last too long. Phenobarbital (grain ¼ to ½) by mouth should be given when the child is able to swallow, and may be repeated at four- or six-hour intervals for twenty-four hours to forestall recurrence.

In *status epilepticus* mild sedation has no effect and convulsions recur when anesthesia is stopped. This requires more heroic measures. Intravenous *sodium luminal* (1 to 3 grains) or *sodium amytal* (1 to 5 grains) may be slowly injected with the needle in place until relaxation occurs. Tremendous doses may be necessary and are comparatively safe. *Lumbar puncture* is at times effective. One per cent *methylene blue* intravenously (10 to 20 cc.) has been used with success; it may be repeated every four to six hours.

It has been advised[7] that diagnostic lumbar puncture be performed invariably at the termination of a convulsion. This seems unnecessary. If definite cause can be found outside the realm of the central nervous system this procedure is not indicated.

CONVULSIONS OF THE NEWBORN PERIOD

INTRACRANIAL HEMORRHAGE OF THE NEWBORN

FREQUENCY.—By all odds the most frequent postmortem finding in stillborn infants and those dying within the first two weeks of life is intracranial hemorrhage. Ford[1] estimates that it is responsible for one third of all neonatal deaths. Sharpe[2] found blood in the spinal fluid in 9 per cent of 100 consecutively born babies; Roberts[3] in 12 per cent.

ETIOLOGY.—In *prematurity* the danger of hemorrhage is

greatly increased, accounting for 39 per cent of all deaths in Hess' series.[4] As corroborative evidence, Glaser[5] demonstrated blood by spinal tap in practically every one of a large series of prematurely born infants. *Difficulty and prolongation of labor, disproportion* between the size of the fetal head and the circumference of the birth canal, whether it be unusual largeness of one or narrowness of the other, the *use of instruments, manual extraction,* and *excessive rapidity of the second stage* predispose to the tears of the tentorium or of the cerebral veins which are responsible for the bleeding. *Hemorrhagic disease of the newborn* must play a definite, though small, role in the production, or at any rate the persistence of intracranial hemorrhage.

SYMPTOMS AND SIGNS.—Since one of the cardinal signs of this disease is *convulsions,* and since it is responsible for such a large proportion of both fatal and the nonfatal illness in the neonatal period, it follows that convulsions within the first two weeks of life are strongly indicative of intracranial hemorrhage. Davison[6] calculates that it is responsible for 55 per cent of the convulsions in the first month; Peterman's[7] figure of 68 per cent is in closer accord with my observation. It must be remembered that convulsions do not occur in all cases. When the hemorrhage is largely supratentorial the symptom-picture is apt to include cyanosis, nervousness (jitteriness), hypertonicity, shrill cry, bulging fontanelle, vomiting, pupillary and cranial nerve palsies, and twitching movements, as well as convulsions. Infratentorial hemorrhage usually gives rise to cyanosis, intermittent periods of apnea with deepening of the cyanosis and slowing of the heart rate, and flaccidity. Convulsions rarely occur in this form unless block supervenes.

In about half of the cases of intracranial hemorrhage seen in the Harriet Lane Home, convulsions were a prominent symptom. In most instances they were generalized; less commonly they were unilateral, or Jacksonian in type, or were limited to tonic contractions of all the muscles for a few seconds (stiffening spells); occasionally twitching movements of the face or extremities were observed, either between generalized convulsions or as the isolated motor symptom of cortical irritation.

PROPHYLAXIS.—To the limited extent that this accident of labor can be prevented, its prevention lies almost entirely in the

hands of the obstetrician. Discussion of this problem does not lie within the province of this article.

In view of Hellman's[8] finding that there is a constant and significant lowering of the prothrombin content in the blood of prematures, coupled with their well-recognized tendency to intracranial bleeding, the administration of *vitamin K* routinely to prematures would seem indicated. The synthetic preparation (2 methyl 1, 4 naphthoquinone in sesame oil) may be given, 1000 units as the first dose, 200 units in each of five successive feedings, at 4-hour intervals. Evidence may accumulate in the future which will warrant similar use of vitamin K prophylactically in all newborn infants.

TREATMENT.—The prime therapeutic agent is *rest*. The infant must not be allowed to attempt to nurse at the breast; feedings should be administered from the bottle, Chetwood syringe, or medicine dropper, in such fashion as to reduce its exertion to the minimum. The head of the bassinet should be kept elevated to decrease venous pressure. *Sedatives* are indicated for jitteriness, twitchings, hypertonus and convulsions. Phenobarbital, grain ⅛ (8 mg.) by mouth every three or four hours may be sufficient. If not, chloral hydrate by rectum, 2 to 3 grains (128 to 192 mg.) every four hours may be effective. Morphine is probably not contraindicated if the dosage is kept small enough, ⅟₆₀ to ⅟₁₀₀ grain (½ to 1 mg.).

Repeated *lumbar puncture* has been vigorously advocated. The facts that the procedure itself is a trying one, both the cramped position and the crying tending to increase intracranial venous pressure, and that in most instances very little fluid can be withdrawn, have convinced many of us that it may be of more harm than good. If the signs of increased pressure are very striking, however, daily punctures should probably be employed.

Craniotomy for the removal of the subdural clot has proved disappointing. Its dangers so heavily outweigh its probable benefits that it cannot be recommended.

TETANY OF THE NEWBORN

FREQUENCY.—This syndrome is rare in the Western world. Bakwin[9] could find only twenty-seven cases in the American literature up to 1939. In the Eastern countries it is more fre-

quent, Chu and Sung[10] being able to report six cases of neonatal tetany in a series of forty-five from the Peiping Union Hospital alone. Hence as a cause of convulsions in the first few weeks of life it ranks very low.

ETIOLOGY.—Chu and Sung[10] found evidence of co-existing *rickets* in four of their six infants. In one the finding was made postmortem, in the other three in vivo by means of the *x*-ray. In two of these, however, the typical radiographic appearance did not become visible until one month after treatment had been instituted. Furthermore, the mothers of these infants had either osteomalacia or had partaken of diets grossly deficient in vitamin D or calcium or both. They feel, therefore, that neonatal tetany is produced by the same factors which cause infantile tetany (*q.v.*). Bakwin[9] maintains, on the contrary, that rickets is not part of the picture in the American cases, and suggests the following etiologic set-up: The maternal parathyroids overfunction in late pregnancy. This depresses the function of the fetal parathyroids. For a few days after birth this parathyroid hypofunction manifests itself by a falling serum calcium in most newborn infants. Under the influence of excessive phosphate ingestion (cow's milk), the calcium is further depressed to a point at which symptoms of tetany appear. It seems probable that neonatal tetany, like that of later life, has several modes of origin.

SYMPTOMS AND SIGNS.—The symptoms may begin at birth, but more commonly become manifest later, from the age of one week on. *Nervousness, twitchings* and *frequent short convulsions* are usual. Carpopedal spasm, the hallmark of infantile tetany, is rare—29 per cent in Chu and Sung's series—and laryngospasm is still rarer. The Chvostek sign is strongly positive with few exceptions, but it is also positive in many normal newborn infants. The Trousseau sign and the peroneal reflex are less frequently elicited, but are also obtainable in some normals. Bass and Karelitz's[11] syndrome of vomiting, high fever, convulsions, carpopedal spasm and positive Chvostek is not the usual picture in the reported cases of hypocalcemic tetany, nor is edema (Shannon[12]) remarked upon by other observers.

   The only reliable, and the pathognomonic sign, is lowering of the serum calcium to 8 mg. per 100 cc. or less.

PROPHYLAXIS.—Adequate vitamin D and calcium intake by the gestating mother should prevent the Eastern form of the disease. In the Western form this seems to play a negligible role, and in at least two instances (Walker[13] and Bloxsom[14]) the administration of percomorph oil to the infant failed to forestall the subsequent development of symptoms.

TREATMENT.—A variety of therapeutic agents can be used with success. After the immediate convulsion has ceased, or has been controlled by ether inhalation, rectal chloral hydrate (grains 2 to 3), or morphine (grain $\frac{1}{60}$ to $\frac{1}{100}$), calcium should be administered. *Calcium chloride* is superior to other salts for oral use, the *gluconate* for intravenous and intramuscular use. An immediate dose of 2 to 4 gm. of the chloride, by gavage if necessary, should be followed by a similar quantity daily, divided into 4 or 6 doses until the serum calcium attains a normal level. If blood calcium estimations cannot be obtained, one may determine this point by the appearance of calcium in the urine by means of. Sulkowitch's[15] reagent, as did Bloxsom.[14] If for any reason medication cannot be given by mouth, or is not retained, calcium gluconate may be given intravenously (5 to 10 cc. of the 10 per cent solution, *i.e.*, 0.5 to 1.0 gm.), or intramuscularly in the same amount once or twice daily. The latter route must not be used frequently because of the danger of tissue necrosis (Tumpeer and Denenholz[16]). It should be stressed that a small dose of calcium may not be adequate and that the gluconate contains considerably less calcium than the chloride. Walker's[13] child received 0.6 gm. of calcium gluconate daily by mouth, plus 0.5 to 1.0 gm. daily intramuscularly, with no clinical improvement and no elevation of the serum calcium. The administration of calcium chloride, 3.3 gm. by gavage, causes prompt cessation of the convulsions.

Bloxsom[14] affected cure in his patient by the oral administration of *dihydrotachysterol* (AT 10). The adequate dose proved to be no less than 15 drops three times a day.

Vollmer[17] has succeeded in producing prompt cure by the use of one massive dose of pure *vitamin D*. He suggests 600,-000 units divided into two successive feedings.

HYPOGLYCEMIA OF THE NEWBORN

FREQUENCY.—Since thus far this condition has been observed only in the offspring of diabetic mothers, and not in all of them, it must be exceedingly rare.

ETIOLOGY.—It is assumed that the pancreas of the fetus attempts to overproduce insulin in an effort to burn the excess of sugar transmitted to it from the maternal blood. Dubreuil and Anderodias,[18] Gray and Feemster,[19] and more recently Smyth and Olney[20] have described tremendous hyperplasia and hypertrophy of the islands of Langerhans in such infants who have died with hypoglycemia shortly after birth. Gray and Feemster reckoned their infant's island tissue to be twenty-four times the normal quantity.

SYMPTOMS.—Many of the babies are oversized and overweight (*macrosomia*). Cyanosis and vomiting, and difficult or rapid shallow respirations are most common; muscular twitching and convulsions occurred four times in Randall and Rynearson's[21] series of eight cases, but not at all in Smyth and Olney's.[20] Indeed, none of the fatal cases mentioned above suffered convulsions.

The *blood sugar*, to be diagnostic, should in the term infant read below 70 mg. per 100 cc. In premature infants this figure is often considerably lower in the absence of hyperinsulinism, so that its interpretation is often doubtful. (Van Creveld[22] found blood sugars in 32 per cent of prematures in the first few days of life below 70 mg., in 16.5 per cent below 60 mg., and in 8.5 per cent below 50 mg.)

TREATMENT.—*Oxygen* should be administered for cyanosis and the child kept in a warmed crib. The specific need is *glucose* which should be supplied by mouth, unless vomiting prevents, by clysis in 3 per cent solution, or by intramuscular injection in 5 per cent or 10 per cent solution as often as necessary. The amount given and the duration of the treatment must depend upon repeated blood sugar determinations.

OTHER CAUSES OF CONVULSIONS IN THE NEWBORN

Tetanus Neonatorum.—The widespread practice of asepsis in the care of the umbilical cord has reduced this formerly common disease to the status of an extreme rarity. The symptoms come on from five to twenty-one days after birth and are

exactly the same as those of tetanus (*q.v.*) of later life. Treatment, too, is the same. The prognosis is very bad. Monsky[23] cured his case by the daily injection of 10,000 units of antitoxin intramuscularly for seven days, and nembutal (grain $\frac{3}{4}$) by rectum every six hours, later every three hours.

Icterus Gravis Neonatorum.—Rarely a child is born jaundiced or develops severe jaundice within the first few days of life. Blood smears usually show an increased number of nucleated red blood cells (erythroblastosis foetalis). Some suffer from one or a series of convulsions within the first weeks. In some, if recovery takes place after several months, cerebral damage becomes manifest by the development of mental retardation, extrapyramidal spasticity, and periodic convulsions (Kernikterus).

Infections.—*Sepsis neonatorum, pneumonia* and *pyelitis* of the newborn are infrequently ushered in by convulsions.

Congenital Heart Disease.—For some reason this condition is rarely complicated by one or more convulsions.

Congenital Cerebral Defect.—This term is used to include the many described types of inborn cerebral malformation: arrested development, atrophic lobar sclerosis, microgyria, primitive convolutional development, as well as porencephaly. In all such infants, convulsions may occur within the neonatal period, although their onset is more often delayed.

DIFFERENTIAL DIAGNOSIS IN NEWBORN PERIOD

Convulsions within the first two weeks of life should arouse the strong suspicion of *intracranial hemorrhage,* on the basis of probability. This is particularly true if they occur within the first week. Those occurring within the second week may be, but are less likely to be, due to hemorrhage. The story of difficult, instrumental or precipitate labor constitutes further evidence. The association of bulging fontanelle, pupillary or cranial nerve palsies, monoplegias, and finally of blood in the spinal fluid clinches the diagnosis. Marked jitteriness, frequent short convulsions with good appetite and seeming wellbeing between, strongly positive Chvostek, Trousseau, and peroneal signs, carpopedal spasm and laryngospasm suggest *tetany of the newborn.* The diagnosis can be made definitely only by the demonstration of hypocalcemia. The known fact

that the mother is diabetic, especially if the infant be unduly large at birth, cyanosis and muscular twitchings suggest *hypoglycemia*. Again the actual proof depends upon estimation of the blood sugar.

The presence of severe jaundice with many nucleated red cells in the blood smear suggests *crythroblastosis*. Finding a *congenital heart lesion* may supply the explanation, but one must remember that in some such conditions murmurs never appear, in others their appearance may be delayed for days or weeks. *Infections* must be searched for even in the absence of fever, as the newborn is notorious for his failure to respond with fever to invading organisms. If no explanation can be found one must suspect the presence of a congenital brain defect although the future course must be awaited before this diagnosis can be considered proved.

CONVULSIONS AFTER THE NEWBORN PERIOD

FEBRILE CONVULSIONS

Particularly between the ages of six months to three years convulsions may occur at the onset of any kind of acute infection. The infection may be a serious one—meningitis, pneumonia or pyelonephritis—but more often than not the seizure announces the beginning of one of the usually benign diseases, tonsillitis, pyelitis or the acute exanthemata. We have been impressed with its frequency at the onset of exanthem subitum.

Wegman[68] has studied the problem experimentally in cats of various ages. He argued that fever itself could not be responsible for convulsions since the former persists but the latter do not recur after the onset; for similar reasons toxemia cannot be held accountable. It seemed to him possible that the younger animal might respond differently to *rapid rise of body temperature* than the adult. He found exactly that. Roughly one half of the kittens and only one sixth of the cats reacted by convulsion to sudden temperature elevation. The more rapid the rise and the higher the temperature elevation, the greater the number of animals who convulsed.

These children usually have but one, sometimes several such attacks at the onset of successive infections. Some, how-

ever, continue to have convulsions later on for no good reason, and have to be classed with the epileptics.

TREATMENT.—Treatment presents no problems. The seizures are usually short and not severe. Cool sponge or cool bath is useful, as is a cool enema. A mild sedative (chloral hydrate grains 3 to 5 per rectum, sodium phenobarbital grain ¼ to 1 subcutaneously) is usually sufficient.

INTRACRANIAL INFECTIONS
PURULENT MENINGITIS

The cardinal signs and symptoms of purulent meningitis are similar, irrespective of the actual infecting organism. Usually the more or less abrupt onset with fever, vomiting, headache (or its counterpart in infants, the shrill "cephalic" cry) and—occasionally—convulsions is followed by the development of stiff neck, positive Kernig's sign, and psychic alterations ranging from delirium to stupor. It is worthy of note that convulsions signalize its onset in only a small percentage of cases. There are a few specific signs which point toward certain etiologic agents.

Meningococcal Meningitis.—Of 334 cases of purulent meningitis seen in the Harriet Lane Home in the years 1930 to 1940, 142 were due to the meningococcus. Convulsions occurred at the onset in only one of eight. The characteristic skin lesions, maculopapular or petechial, were present in one third to one fourth of all cases. When present this rash constitutes a great aid in differential diagnosis. A fulminating form, in which death occurs in a few hours, and a low-grade chronic form, in which vomiting, low-grade fever, and slowly developing hydrocephalus may be the only symptoms (chronic basilar meningitis), are less frequently encountered.

TREATMENT.—After several years of comparative experience, both Hodes* and Walker† have practically given up anti-meningococcus serum in favor of *sulfanilamide.* Hodes offers in justification of this stand the comparative mortality rates in a large series of 24 per cent with serum and 15 per cent with the drug. The author is in full accord with this decision.

Treatment should be begun with one or two hypodermocly-

* Superintendent of the Sydenham Hospital, Baltimore, Maryland.
† Resident Pediatrician of the Harriet Lane Home, Baltimore, Maryland.

ses of 1 per cent sulfanilamide in one sixth molar sodium lactate, the first clysis containing the computed daily dose. Depending upon the severity of the disease the daily dose should be reckoned at 1 to 1½ grains per pound of body weight. Thereafter the daily dose should be divided into six fractions and given by mouth with an equal quantity of sodium bicarbonate. If vomiting is persistent, or if the sulfanilamide level —which should be determined daily—falls below 10 mg. per 100 cc., parenteral administration should be continued. If the clinical response is satisfactory, as it almost invariably is, no further spinal puncture after the original diagnostic one need be performed for three or four days or more.

If *serum* is used, 15 to 30 cc. should be injected intraspinally, provided that much fluid has been withdrawn, twice in twenty-four hours for two or three days, and every twenty-four hours thereafter until the fluid becomes sterile.

Pneumococcal Meningitis.—This infection is the next most frequent offender, accounting for sixty-two of the 334 cases. It should be suspected when meningeal signs appear during the course of pneumonia or pneumococcal otitis media. Its inception may be explosive, with convulsions, delirium, stupor and palsies, or it may pass unnoticed for days, only to be suspected by increased fontanelle tension or the failure of the original disease to improve. Now that specific treatment is available, and is more effective when begun early, it is imperative that patients with pneumococcal infection be observed closely throughout their illnesses for signs suggesting this complication.

TREATMENT.—This should consist of both *sulfapyridine* and *serum*. The drug blood level should be rapidly elevated to 12 to 15 mg. per 100 cc. and maintained there. One or two intravenous doses of sodium sulfapyridine, 0.05 gm. per kilogram (about ⅛ grain per pound), should be followed by a daily oral dose of about 0.3 gm. per kilogram (2 grains per pound), divided into four fractions. Rabbit antiserum of the specific type should be injected intravenously (100,000 units daily until the temperature falls) and intrathecally (10 to 20 cc. daily).

B. Influenzae Meningitis.—This infection occurred fifty-two times in our series. In the majority there are no dis-

tinguishing signs, but the presence of acute arthritis or peri-arthritis preceding the meningeal infection should arouse suspicion.

TREATMENT.—This is exactly that of pneumococcal meningitis, except that specific horse serum (Silverthorne and Brown[24]) or rabbit serum (Alexander[25]) against the B. influenzae is used. Daily intravenous and twice daily intrathecal injections for the first two or three days should be followed by daily intrathecal treatments until three successive negative spinal fluid cultures have been obtained. One can expect a reduction in mortality from the nearly 100 per cent of the non-specifically treated group to 70 per cent or less.

Streptococcal Meningitis.—Less common than the above three, this infection occurred twenty-two times in the series of 334. Almost invariably it follows neighborhood infection, such as otitis, mastoiditis or sinusitis. Treatment, after proper surgical attention to the original focus, is exactly as described for meningococcal meningitis.

TUBERCULOUS MENINGITIS

ETIOLOGY.—Dissemination of tuberculosis from a distant focus, in many cases previously unrecognized, is responsible for the disease. Infants from one to three years of age are most frequently affected, but no age-group is immune. Negro infants are more often attacked than white.

SIGNS AND SYMPTOMS.—Gradually increasing drowsiness is the outstanding symptom. Convulsions, with headache and vomiting, may mark the onset, not to recur until the terminal stage. Stiff neck, Kernig's sign, retraction of head may not appear early. Automatic purposeless movements, pupillary or facial palsies, and transient monoplegias or hemiplegia may be found. The *tâche cérébrale* (dermatographia) is usually marked. Deep stupor, strabismus and irregular respirations mark the late stages. Tubercles are rarely seen in the retina and the tuberculin test is strongly positive. The spinal fluid is under increased pressure, water clear—but forms a pellicle upon standing and contains 50 to 1000 cells, usually about 200, per cu. mm. Globulin is definitely increased. With sufficient effort tubercle bacilli can be found in the fluid in most cases.

TREATMENT.—No effective treatment is known.

THE MENINGO-ENCEPHALITIDES

Primary Virus Infections.—ETIOLOGY.—These diseases, with many similarities and few distinguishing characteristics, are caused by filtrable viruses. The group includes *poliomyelitis*, the *encephalitides* (lethargic or von Economo's, St. Louis, equine, Australian X, etc.), and *lymphocytic choriomeningitis*. No age-groups are immune, although the different viruses affect certain ages preferentially.

SYMPTOMS AND SIGNS.—Fever, headache, vomiting, drowsiness and stiff neck or back are common at the onset of most of them. Convulsions are frequent early in the course of the encephalitides, less so in poliomyelitis and choriomeningitis. Single or multiple muscle-group flaccid paralysis is peculiar to poliomyelitis. Paucity of meningeal signs, and oculomotor palsies are the rule in lethargic encephalitis; rapid development of stupor in the St. Louis form. The symptoms of choriomeningitis are apt to be exceedingly mild.

The *spinal fluids* in all show pleocytosis, of 20 to 500 cells. In choriomeningitis practically all cells are lymphocytes, in the others polymorphs predominate the first few days, to give way to mononuclear cells thereafter. Globulin is usually present in small quantities. Pellicle seldom forms. Culture of the fluid is sterile.

Differential diagnosis within the group can only be affected by animal inoculation, in the acute stage, and by the demonstration of neutralizing substances in the individual's blood after the attack is over. For details the reader is referred to Rivers' exhaustive book on the subject.[26]

COURSE.—The severity of the meningo-encephalitides varies greatly both with the particular disease and in individual cases within each group. In general it may be said that the febrile course lasts from five to twenty days, being shortest in choriomeningitis, longer in poliomyelitis and the St. Louis form of encephalitis, and longest in lethargic encephalitis. Sequelae follow the same pattern, choriomeningitis leaving none, St. Louis encephalitis a few (Hempelmann,[27] Zentay and Basman[28]), lethargic encephalitis many.

TREATMENT.—Except for the use of sedatives, and repeated spinal drainage for the relief of the symptoms of increased intracranial pressure, there is no useful therapy.

Postinfection Encephalitis.—During the course of, or shortly following, an attack of *measles, influenza, mumps, vaccinia* or *varicella* an encephalitis may develop clinically indistinguishable from the above. Pathologically it differs in that, in the first group, foci of round cell infiltration, in the second, of perivascular demyelinization are the characteristic features. The onset is usually signalized by one or more convulsions, except in mumps, and fever, headache and vomiting. Meningeal signs are not prominent, again except in the case of mumps. The spinal fluid is identical with that of the first group.

Encephalitis following mumps leaves no sequelae, that following measles many.

Treatment is ineffective.

Convulsions During Pertussis.—Various pathologic processes, or none, may be found in the brains of children who have died with convulsions complicating pertussis. Multiple petechial hemorrhages are common, and may represent cause or effect. Rarely one large intracerebral hemorrhage is the unquestioned cause. More often extensive loss of ganglion cells, involving the convolutional cortex and even the central gray matter, without inflammatory change, is found. Zimmerman[29] presents excellent evidence to prove that this, too, is the effect, not the cause, of the convulsions. Powers[30] has demonstrated the co-existence of infantile tetany in a fair proportion of cases.

Treatment is entirely symptomatic.

INTRACRANIAL VASCULAR DISEASE

Subdural Hematoma, Pachymeningitis Hemorrhagica Interna.—Etiology.—These two conditions are being considered together because their similarities heavily outweigh their differences. The first is consequent upon trauma to the head, but symptoms may not put in appearance until weeks or months after the injury. In the second there is rarely a definite history of trauma, and frequently enough there is associated disease which accounts for a tendency to bleeding. *Scurvy* is present in a large number of cases (Ingalls[31]), as is *congenital syphilis*. In this type, too, there seems greater tendency for the lesion to expand and toward spontaneous repetition of hemorrhage, corresponding to the greater vascularity of

the membranous envelope which encloses the extravasated blood.

SIGNS AND SYMPTOMS.—The spontaneous form is commonest between the ages of six months and two years. Chief symptoms are failure to eat properly and gain weight normally, vomiting, periodic convulsions, enlargement of the head, and failure to develop mentally. The fontanelle becomes full and tight, the veins of the scalp distended, and subhyaloid hemorrhages often appear in the fundus. Diagnosis is confirmed by finding clear spinal fluid—seldom the hematoma ruptures into the subarachnoid space and the fluid is xanthochromic and contains crenated red blood cells—and a quantity of fluid containing fresh or old blood by subdural puncture at one or both lateral angles of the fontanelle.

In older children the disease is usually traumatic in origin and the signs are those of a space-consuming, irritating mass over the cerebrum. Diagnosis can be confirmed by subdural puncture after trephining.

TREATMENT.—Associated disease must be treated specifically. If scurvy is present, or suspected, large doses of orange juice or pure vitamin C are indicated. Repeated *subdural taps* with withdrawal of as much of the fluid mass as possible may prevent excessive cortical damage. Since patients treated in this conservative fashion frequently develop serious mental retardation and epilepsy, *operative removal* of the neomembrane should be seriously considered. Large size and rapid refilling of the emptied sac are indications for operative interference (Ingraham and Heyl[32]).

Subarachnoid Hemorrhage.—ETIOLOGY.—Sands[33] lists eight possible causes for subarachnoid hemorrhage. In children it is probable that only one is of any real importance, *rupture of an intracranial aneurysm,* situated in or near the circle of Willis. Rarely the aneurysm will be found to be infective in origin, but most are congenital.

SIGNS AND SYMPTOMS.—Symonds,[34] Sands[35] and Albright[36] have described the clinical picture in detail. It includes a sudden apoplectic attack, preceded usually by severe headache, occasionally by convulsion, and followed by pain in the back of the head and neck and *stiffness of the neck and back.* The latter, the signs of blood leakage, may have followed a

period of supraorbital pain, with various cranial nerve palsies, chiefly affecting the eye, signifying pressure upon these nerves. Retinal hemorrhages are not rare. Hemiplegia is uncommon. Low fever and leukocytosis are present and may be confusing. Massive albuminuria in children is exceptional. Attacks may be repeated at long intervals but single attacks are probably the rule.

The spinal fluid is uniformly bloody, with xanthochromic supernatant liquid.

TREATMENT.—Absolute *rest* is essential. This should be continued until one is fairly sure that complete healing has taken place—a period of roughly six weeks.

Operation is indicated only when the location of the aneurysm can be determined, by the presence of pain or persistent palsy. Occasional excellent results have been obtained (Dandy).[37]

Cerebral Sinus Thrombosis.—ETIOLOGY.—There are two distinct groups of illness which predispose to thrombosis of the cerebral sinuses. The *infectious* group includes the thromboses resulting from the spread of infections from neighboring structures (mastoids, meninges, sinuses, scalp), usually into the lateral or cavernous sinuses, and thrombosis occurring in the course of septicemia. This type seldom, if ever, gives rise to convulsions (except in the rare instance of retrograde spread of the thrombus into the superior longitudinal sinus), hence its description does not lie within the scope of this article. The *primary* form, previously called "marantic," invariably involves the superior longitudinal sinus. Byers and Hass[38] consider increased viscosity of the blood and slowing of the rate of blood flow the most important causative factor.

SIGNS AND SYMPTOMS.—It occurs shortly after the onset— several days to several weeks—of a disease in which severe dehydration is prominent. Usually this is an acute diarrheal disease; in the Harriet Lane Home a recent case followed a burn. There may be no symptoms whatsoever. If the thrombus is extensive, convulsions mark the onset, and generalized rigidity or hemiplegia may develop. The retinal veins become engorged and hemorrhages may be seen; the scalp veins may become distended and themselves thrombosed. The spinal fluid may be normal or there may be slight increase of red

blood cells, white blood cells and protein; rarely, the fluid is xanthochromic or grossly bloody. Death is the usual outcome; recovery may be followed by mental retardation, epilepsy and spastic paralysis, with pneumo-encephalographic evidence of cortical atrophy (Byers and Hass[88]).

TREATMENT.—Infected thrombi of the lateral sinuses should be treated *surgically*. The ideal treatment consists in tying off the internal jugular vein in one stage, and removing the thrombus from the lateral sinus in another. Specific chemotherapy should depend upon the infecting organism (see Purulent Meningitis).

Primary thrombi cannot be successfully attacked surgically. Treatment is purely *preventive;* severe grades of dehydration in illnesses in which vomiting and diarrhea are prominent, and in severe burns, must not be allowed to develop. It has been remarked that this accident seems to be increasing in frequency, its rising incidence paralleling the increasing use of intravenous therapy and particularly the scalp vein route. No clear-cut demonstration that the relationship is causal has been made.

Strümpell-Marie Encephalitis (Polio-encephalitis).— ETIOLOGY.—In 1927 Ford and the author[39] reported a fatal example of this syndrome and carefully reviewed a large series of nonfatal ones. Thrombosis of the middle cerebral artery was found postmortem in their case and they concluded that intracranial vascular thrombosis, rather than encephalitis, was the most likely cause in all. Several of Ebb's[40] cases were indistinguishable clinically; postmortem he found thromboses of venous sinuses.

SIGNS AND SYMPTOMS.—There is sudden onset of convulsions, with high or rapidly rising fever. Frequently a mild upper respiratory infection has been present. The convulsions are long, severe, usually generalized and often repeated. They may be one-sided. Following their cessation fever rapidly subsides and complete hemiplegia is noted.

The spinal fluid throughout remains normal. Unlike the transient hemiplegia which occasionally follows a convulsion from any cause (Davis[41]) it clears but slowly and imperfectly, the leg first and perhaps entirely, the arm later and only partially. Epilepsy and mental deterioration are common sequelae, developing in 70 per cent of the cases.

TREATMENT.—No treatment is of avail in the acute stage. Orthopedic measures should be directed toward the residual hemiplegia.

THE TETANIES

ETIOLOGY.—The clinical picture known as tetany can be brought about in several different ways. Most commonly it develops in association with rickets, as a result of an insufficiency of vitamin D in the diet and of inadequate exposure to effective sunlight. This combination of circumstances leads to a fall in the phosphorus content of the blood serum, and less frequently to a similar drop in its calcium content. When the calcium falls below 8 mg. per 100 cc. tetany becomes manifest. This form is known as *infantile tetany*. It is one of the most frequent causes of convulsions between the ages of six months and two years, less so before and after.

The internal secretion of the parathyroid glands is concerned with the maintenance of the proper calcium level in the blood by regulating its flow to and from the bones. When these glands are congenitally absent, or are removed at operation, as has rarely occurred, the serum calcium again falls below the critical level and *tetania parathyreopriva* results.

Bakwin[9] believes *neonatal tetany* to be the result of temporary hypoparathyroidism (see above).

Prolonged *steatorrhea,* such as occurs in celiac disease and in pancreatic insufficiency, may lead to hypocalcemia and tetany, since vitamin D, being fat-soluble, is not absorbed from the intestine in sufficient quantity.

Hyperventilation brings about the same symptoms. The mechanism involved consists of the production of alkalosis by loss of CO_2, with probable reduction of ionizable calcium. Hyperventilation occurs as a symptom in the postencephalitis syndrome, also more rarely as an hysteric phenomenon (see Grant and Goldman[42]).

Gastric tetany results from prolonged vomiting from any cause. Loss of the chloride ion is responsible for the development of alkalosis (MacCallum[43]). We have seen a CO_2 combining power of 120 volumes per cent result from the persistent vomiting of pyloric stenosis in an infant of five weeks.

SIGNS AND SYMPTOMS.—Regardless of the cause, the symptom-complex of tetany is the same. Characteristic are *convul-*

sions, *carpopedal spasm*, and *laryngospasm*. The convulsions are usually generalized, very rarely unilateral. They are often repeated. They are not followed by the stuporous period so usual after those from other causes. They are more common, and carpopedal spasm less so, in infants than in older children with tetany. Signs of increased nervous irritability can usually be elicited. *Chvostek's* sign (facial phenomenon), *Trousseau's* sign (production of carpal spasm by tourniquet above the elbow), *peroneal* sign (foot-jerk by tapping just below the fibular head), and *Erb's* sign (increased electrical excitability) are generally positive. Rickets can almost always be demonstrated, radiographically if not clinically, in infantile tetany. Positive diagnosis is dependent upon the demonstration of hypocalcemia (in the infantile, neonatal, and parathyreoprival forms) or of alkalosis.

TREATMENT.—For neonatal tetany, see above.

The treatment of *infantile tetany* is similar to that of the neonatal form (*q.v.*). One large dose of *calcium chloride* (2 to 4 gm., 30 to 60 grains) should be given by mouth, by gavage if need be, and followed by a similar dose daily, divided into 4 or 6 fractions. After the serum calcium has attained a normal level it may be tapered off. *Vitamin D* should be administered, but only after an interval of two or three days, since a rapid rise of the serum phosphorus may cause a coincident rapid fall in the serum calcium and thus produce relapse.

(For alternative methods of treatment, one massive dose of vitamin D, parathormone, dihydrotachysterol, see Neonatal Tetany.)

Dihydrotachysterol is the medication of choice in parathyreoprival tetany. Rose and Sunderman[44] recommend, for adults, 5 to 10 cc. daily for the first three or four days, followed by a maintenance dose of 2 to 6 cc. weekly. The actual amounts needed must be determined by repeated estimations of the serum calcium.

Steatorrheic tetany is treated at first as is infantile tetany. Calcium therapy should be followed by ultraviolet light therapy instead of, or in addition to, oral vitamin D.

The treatment of *hyperventilation tetany* must be directed toward the cause of the hyperventilation itself.

Intravenous and subcutaneous sodium chloride in large

quantities will usually relieve *gastric tetany*. If it does not, 1 to 4 cc. of one-tenth normal hydrochloric acid may be added to a slowly flowing continuous intravenous drip of isotonic sodium chloride.

HYPOGLYCEMIA

ETIOLOGY.—A blood sugar level of below 70 mg. per 100 cc. is usually considered hypoglycemia. Children, however, seem to be more tolerant than adults, symptoms seldom occurring until a level of 60 mg. or less, and often not until 40 milligrams or less, is reached. An *overdose of insulin* is the commonest cause. Certain liver-damaging *poisons*, particularly chloroform, phosphorus and hydrazine, regularly produce it. Inability to transform glycogen into glucose, as happens in *von Gierke's disease*, or *hyperinsulinism* because of maternal diabetes or due to pancreatic adenoma may be responsible. Not fully understood disorders of the carbohydrate-regulating mechanisms of the pituitary and adrenal cortex may be involved. Finally, there is a group for which no explanation can be found, the so-called *spontaneous hypoglycemia*. In two patients who died of this condition, Josephs[45] found in both increased fat accumulation in the liver, in one diffuse scarring of the liver.

SIGNS AND SYMPTOMS.—Children who have received an overdose of insulin usually become *drowsy, dizzy* and *ataxic*. They may see double. A state of *shock* with rapid weak pulse, profuse perspiration, soft eye-balls, occasionally convulsions and coma may follow.

The picture in neonatal hypoglycemia has been described.

Repeated convulsions may occur in von Gierke's disease.

Josephs' spontaneous hypoglycemics suffered from recurrent bouts of *vomiting* and *acidosis*, with or without convulsions. Usually the bouts were precipitated by upper respiratory infections, but fasting for one or two meals was sufficient to initiate the attack in others. Indeed, the characteristic time for vomiting to begin was in the early morning, after the longest normal fasting period of the twenty-four hours. Convulsions occurred in seven of his ten cases.

Seale Harris[46] has reported a number of cases of recurrent convulsions (and one of narcolepsy) in which he found hypoglycemia, the patients allegedly being cured by partial pan-

createctomy. We have seen one similar case in the Harriet Lane Home, a five-year-old boy with recurrent convulsions, usually in the early morning. Unlike those of Josephs' group, these convulsions were unaccompanied by any evidence of infection, vomiting or acidosis; indeed, they were indistinguishable from those of idiopathic epilepsy. Blood sugars of 47 and 52 mg. were discovered on two occasions.

TREATMENT.—*Glucose* is the specific remedy for hypoglycemia. In insulin overdosage eating an ounce or two of chocolate or drinking a glass of sweetened orange-juice often suffices. If unconsciousness renders oral administration impossible, 5 per cent glucose may be given in retention enemata, or 5 per cent or 10 per cent glucose intravenously.

For those children with recurrent vomiting or convulsions due to spontaneous hypoglycemia, the addition of intermediate nourishment high in carbohydrate seems to be enough. The snack at bedtime is the most important one; at times it is advisable to awaken the child at midnight or later for a cup of chocolate or the like. Whatever error of metabolism is responsible for their unduly labile blood sugar level seems to disappear with advancing age.

Should these simple measures not suffice to keep the child free from distressing attacks, hyperinsulinism should be suspected. A mildly ketogenic diet may be tried (Harris[46]); if it is of no avail, exploration of the pancreas should be seriously considered.

CEREBRAL POISONINGS

Lead Encephalopathy.—ETIOLOGY.—No age is immune to lead poisoning. The younger the individual, however, the more susceptible he is to the cerebral form of the disease. The gastro-intestinal and peripheral nerve symptoms are apt to be lacking or of minor importance in infants and young children.

Lead may be absorbed either by ingestion or inhalation. Young infants ingest it from breasts upon which lead-containing ointments or lead nipple shields have been used. Babies of six months or more chew the paint from their beds, play pens, or the porch railing. Children with perverted appetites (pica) will eat anything. Williams[47] has reported an epidemic in Baltimore caused by the use of discarded battery casings,

impregnated with lead salts, as a fuel. Relatively few cases occur in the first six months, most from the ages of one to three years, and a few thereafter.

SIGNS AND SYMPTOMS.—Of seventy-seven patients with lead poisoning reported by McKhann and Vogt,[48] forty-five had encephalopathy. Convulsion is the usual presenting symptom, and is severe, protracted and prone to repetition. Associated are *vomiting*, often projectile, *mental change, delirium, stupor* or *coma*. Prodromal symptoms often include abdominal pain and constipation. Examination usually reveals pallor, elevation of blood pressure and choked disks. Lead line is infrequently present. The blood shows anemia and often many stippled red blood cells. x-Ray of the extremities of young, rapidly growing children shows the characteristic lead line, a broad, opaque band at the epiphysis (Park, Jackson and Kajdi[49]). The spinal fluid is clear, under increased pressure and contains a quantity of protein out of proportion to the slight mononuclear cell pleocytosis (30 to 250 per cu. mm.). Glycosuria is often present. Spectroscopic examination of the blood confirms the presence of lead, quantitative examination reveals a lead concentration of 0.05 mg. per 100 cc. or more.

Eleven of McKhann and Vogt's[48] series of forty-five patients died of the acute cerebral edema. Of the thirty-four survivors, twelve developed permanent sequelae in the form of mental retardation, cerebral atrophy, epilepsy, blindness, etc. Hence the prognosis is very grave.

TREATMENT.—Aub[50] was the first to suggest a specific method calculated to hasten the deposition of lead into the bones, and another to de-lead the bones. Unfortunately, the first—large doses of calcium and vitamin D—has not proved very effective, and the latter is considered dangerous. Shelling[51] advised the substitution of large doses of *phosphorus* (2 to 4 gm. of disodium phosphate daily, in addition to large quantities of milk) for the calcium of Aub's regime, and proved the method effective in *preventing* lead poisoning in rats. Its therapeutic effect is doubtful, but it should probably be tried *faute de mieux*.

Surgical decompression has been attempted in several clinics with the dual purpose of preventing death from acute cerebral edema, and of minimizing the cerebral damage from

the greatly increased intracranial pressure. Haverfield, Bucy and Elonen[53] have recently reported a series of five such attempts. Two children died quickly, one of severe cerebral herniation through the decompression wound, one was left slightly retarded mentally, two had no sequelae. The series is obviously small, but apparently not too much should be expected from this procedure.

Tetanus.—ETIOLOGY.—The symptoms of tetanus arise from the diffusion of the soluble exotoxin of Clostridium tetani from a wound infected with that organism. In perhaps one fourth of all cases the original wound cannot be discovered. Puncture wounds and compound fractures are the most dangerous. Tetanus neonatorum, a rarity now, arises from umbilical infection.

The toxin is not carried to the central nervous system via the peripheral nerves. Part of it diffuses into the neighboring muscles, affecting the myoneural end plates, the remainder is absorbed into the circulation whence some of it is removed and fixed by the central nervous tissue (Abel[53]).

SIGNS AND SYMPTOMS.—After an incubation period of from five days to three weeks or more, local tetanus may appear, consisting of stiffness, with or without pain, of the muscle groups nearest the site of infection. This stage occurs but rarely in children. General tetanus develops, with gradually increasing rigidity of all the striated musculature and periodic convulsions. Characteristic manifestations of tetanic rigidity are *lockjaw* (trismus), the *risus sardonicus*, and *boardlike consistency* of the abdominal wall. Characteristic features of the spasm are the failure to lose consciousness, their painfulness, and the fact that they are initiated by such slight stimuli as a sudden noise, or a touch. The blood and spinal fluid are normal.

TREATMENT.—*Prophylaxis.*—Evidence has been accumulating indicative of the value of active immunization by means of tetanus *toxoid*. Many of us were fearful of it because of that percentage of individuals who failed to respond to it by the formation of antitoxin, and because of our comparative inability to identify the recalcitrants. There is no skin test to demonstrate immunity. Recent reports (see Gold[54]) show that proper dosage confers immunity on nearly all of the in-

oculated. The author does not hesitate to advise its use in all babies. Combined with diphtheria toxoid, three doses should be given, the first at six months, the others after intervals of six weeks to two months. For the third injection intranasal instillation of *Topagen* may be substituted, 0.1 cc. in each nostril twice. This procedure effectively raises the antitoxic titer of the blood shortly after the second injection, but it falls to a low level within several months. It is only necessary, however, to inject an additional dose of toxoid whenever it is desired to raise the titer again to a high level.

The traditional 1500-unit dose of *antitoxin* is not sufficient protection against tetanus if there has been an extensive wound, especially compound fracture. A second dose after five or six days is advisable in these cases.

Therapy involves decision as to the amount and route of the original dose of antitoxin, the frequency and quantity of further doses, if any, and the type of antispasmodic drug to be employed. Firor[55] in 1939 advised an initial dose of 50,000 units intravenously, 10,000 units to be infiltrated about the wound, followed by thorough débridement after one hour; thereafter 5000 units of antitoxin intramuscularly daily. Since then he[56] has demonstrated that in experimental animals results are appreciably better in not too severe cases when the original injection is intrathecal, and he is now advocating that procedure in humans.

Dietrich[57] makes an excellent argument against the use of antitoxin intravenously and intrathecally. He presents evidence, in an unfortunately small series, that these injections are responsible for most of the deaths within the first thirty-six hours of admission. None of his patients treated by intramuscular injection alone died!

Until further data become available a middle stand should be taken. An initial dose of 30,000 to 50,000 units, one quarter about the wound, one quarter intravenously and one half intramuscularly should be sufficient. Intramuscular injections of 5000 units daily, or every other day, should maintain a high titer until symptoms begin to subside. Intrathecal and second intravenous injections should probably be avoided.

Débridement should be performed, after the initial treatment.

The *sedative* employed should be antispasmodic and should induce a constant light sleep from which the patient can readily be aroused every hour or two for fluids. Firor[35] prefers paraldehyde, chloral, or ether in oil, and believes avertin to have too depressant an effect, especially upon the swallowing reflex. Dietrich[57] prefers seconal (3 to 4 grains every four hours for a five-year-old). Nembutal (¾ grain every three hours) has been used successfully in the treatment of tetanus neonatorum (Monsky[23]).

If the attempt to administer fluids causes too severe spasms, one may resort to a Murphy drip or a continuous intravenous drip.

Other Poisons Producing Convulsions.—The list of poisons, headed by strychnine, which can produce convulsions is too long for any adequate discussion of their symptomatology and therapy to be included in this communication. The reader is referred to the special chapters in Abt's or Brenneman's Systems of Pediatrics.

CEREBRAL EDEMA

Lead poisoning causes convulsions by the production of edema of the brain. It has been discussed under the alternative heading of "Poisons."

Acute Nephritis.—In a not inconsiderable number of children with acute hemorrhagic nephritis, rising blood pressure, vomiting, headache, drowsiness and convulsions mark the development of complicating cerebral edema (Blackfan[58]). The mechanism is not fully understood. *Treatment* should be instituted for steadily rising blood pressure, especially if vomiting and drowsiness appear. Magnesium sulfate by mouth (30 to 90 cc. of the 50 per cent solution every four hours) may suffice. If not, the 25 per cent solution can be given intramuscularly (5 to 10 cc. every four or six hours) until the symptoms abate. The intravenous route has also been advocated but is generally regarded as too dangerous.

CONGENITAL CEREBRAL DEFECTS

In many of the inborn defects of the brain there is no predisposition to recurring convulsions. The Mongolian idiot, for instance, practically never suffers them. In certain varieties, however, they are extremely common.

Microcephaly.—In microcephaly the brain fails to grow at the normal rate, hence the skull fails to expand. It is a common misconception that the failure of the brain to grow is due to premature synostosis of the skull. The characteristic small size, with backward sloping forehead and flat occiput, makes their recognition unavoidable. Convulsions occur in one third to one half of microcephalics.

Treatment of choice is phenobarbital, ¼ to ½ grain three times daily, or more if necessary. Surgery should not be considered.

Hydrocephalus.—This condition is due either to congenital or acquired obstruction at the aqueduct or at the foramina of Luschka and Magendie, rarely to overproduction or underabsorption of cerebrospinal fluid. The rapidly enlarging head, tense fontanelle and rolled-down eyes are unmistakable.

Treatment is expectant or surgical. In some instances spontaneous arrest occurs. Dandy has devised the operations of third ventriculostomy for the obstructive type, and choroid plexectomy for the communicating form. Cure results in a few instances.

Tuberous Sclerosis.—Tuberous sclerosis is an exceedingly rare condition in which recurrent convulsions are associated with skin lesions known as *adenoma sebaceum,* and mental defect.

Spastic Diplegia.—The origin of congenital spastic diplegia has given rise to much argument. Ford[1] has adduced excellent evidence to prove that only rarely can it be a result of intracranial hemorrhage at birth; the great majority who come to autopsy reveal cerebral malformations which must be congenital. Spasticity may be present from birth, but more often it is not noticed until the latter half of the first year. Usually the legs are affected more than the arms, and the adductors more than the abductors, so that when the patient is held up the typical scissors position is assumed. Physical development is always, mental development often, retarded. Mild microcephaly is not uncommon, recurrent convulsions frequent.

Treatment is largely orthopedic. Phelps[59] believes a great deal can be done by means of re-education, studied relaxation and physiotherapy. Convulsions are best treated by phenobarbital.

Epilepsy.—ETIOLOGY.—It seems justifiable to classify epilepsy under the general heading of "Cerebral Defect" although there can be no doubt that some cases are acquired rather than inborn. In the Harriet Lane Home series of 254 children beautifully analyzed by Wilkins,[60] sixty had definite neurologic lesions, and microcephalics and spastic diplegics were not included in his study. Eighty-two more were mentally defective, leaving but 111 with possibly normal brains. Had pneumo-encephalography been practiced, or surgical exploration, surely a large percentage of these would have been found to have lesions. Finally, the demonstration of abnormal electrical waves (Gibbs[61]) not only in the epileptic, but in one or both parents (in 94 per cent) speaks eloquently for some abnormal anatomic substrate. Penfield[62] has found a great variety of lesions in the exposed cortices: neoplasms, scars, focal microgyria, cysts, congenital malformations, etc. Ney[63] lays great stress upon cortico-cerebral scars. The mechanism of the production of periodic convulsions is not clear.

SIGNS AND SYMPTOMS.—Recurrent major *convulsions,* minor *motor attacks,* or episodes of *petit mal* constitute the clinical picture. There may be associated mental or motor defect, as described above. Encephalography with air may or may not reveal areas of cortical atrophy. Electro-encephalography will reveal the waves characteristic of grand or petit mal.

COURSE.—Attacks may begin at any time in infancy or childhood. They may be repeated several times daily or only once annually. Spontaneous remissions occurred in about one quarter of Wilkins' cases, more frequently in the mild forms than in the severe. Some become worse, mental deterioration progresses and death occurs in status epilepticus.

TREATMENT.—Remission and cure may follow the use of various anticonvulsants. The use of *bromides* (5 to 15 grains t.i.d.) has been largely given up because of the frequency of skin and nervous effects. *Phenobarbital* (grain ¼ to 1 b.d. or t.i.d.) is occasionally effective. *Dilantin sodium* has given somewhat better results than the other anticonvulsants and does not produce the undesirable side-effect of sluggishness common to the others. In most series (see Merritt and Putnam,[64] and Fetterman[65]) seizures were stopped or sharply re-

duced in from 30 to 50 per cent. On the other hand its side-actions, dermatitis, stomatitis and severe gastro-intestinal upset, are frequent and distressing. They can be kept to a minimum if the dosage is begun low and gradually increased (½ grain t.i.d. one week, 1 grain t.i.d. second week, 1½ grains t.i.d. thereafter).

Failing favorable result from drugs, *ketogenic diet* should be tried. Two to five days of preliminary starvation, until ketosis, as evidenced by the appearance of strongly positive acetone and diacetic acid tests, is well established are followed by a ketogenic diet of sufficiently high ratio to maintain ketosis. This is usually 4:1 in terms of calories from fat to the sum of calories from protein and carbohydrate. A generous allowance of protein (2 gm. per kilogram) should be allowed for growth needs, and the vitamins should be included in abundance. This diet is maintained for two years, and thereafter gradually tapered off. Cessation of seizures may occur immediately or gradually over a period of months, or not at all. Wilkins[60] obtained 26.6 per cent cures, Helmholz and Keith[66] 36 per cent, Talbot[67] 25 per cent.

Surgical exploration should be considered (*a*) if the seizures are very frequent or becoming more so, (*b*) if drug therapy and ketogenic diet have proved unavailing, and (*c*) if by means of focal attacks, pneumo-encephalography or electro-encephalography the irritative focus can be localized. Penfield[62] and Ney[63] have reported excellent results from various operative procedures.

DIFFERENTIAL DIAGNOSIS

The arrival at an accurate diagnosis in a convulsing child depends upon the correct interpretation of many factors, some in the child's history, others in his examination, and of the laboratory procedures. Much of this is elementary.

Periodically *recurrent convulsions* suggest idiopathic epilepsy, rarely hypoglycemia. Associated mental retardation with recurrence suggests microcephaly, cerebral agenesia, spastic diplegia, rarely tuberous sclerosis.

Failure to lose consciousness during the attack indicates tetanus or strychnine poisoning. Rapid return of mental alertness is suggestive of tetany.

Most attacks are generalized. When *unilateral*, vascular

thrombosis, tumor or polio-encephalitis should be suspected, but tetany and lead can be responsible. When focal or Jacksonian, subdural hemorrhage, tumor or abscess is not unlikely.

Absence of fever will rule out infections. Its *presence* may indicate intracranial or meningeal infection, or a nonspecific one. It may also be present in tetany (due to the precipitating infection), and in subarachnoid hemorrhage.

Choked disks do not necessarily imply tumor. They may be found in lead encephalopathy and the cerebral edema of acute nephritis.

Hypertension similarly may be discovered in intracranial tumors, lead poisoning and acute nephritis.

Normal spinal fluid rules out meningeal infections, the encephalitides, and lead encephalopathy. It is customarily found in subdural hematoma, polio-encephalitis and tetanus.

Clear spinal fluid with a moderate increase in cells and great increase in globulin strongly suggests lead encephalopathy. Clear fluid with increased cells and slight globulin increase suggests the meningo-encephalitis group. Proportionate cellular and globulin increase is usual in tuberculous meningitis. Turbid fluid means purulent meningitis.

Bloody spinal fluid, if faulty technic can be excluded, suggests intracranial hemorrhage of the newborn, ruptured intracranial aneurysm, occasionally dural sinus thrombosis, or rupture of a subdural hematoma.

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MENTAL DISTURBANCES IN ADOLESCENTS

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Every day of man's life constitutes a transition between yesterday and tomorrow, converging in the intrinsic values of the moment. The past and present continue to shape and modify directions and goals, mostly through quiet evolution, sometimes in lively spurts.

Youth a Period of Transition.—One of the liveliest spurts occurs during adolescence, at a time when a person is no longer a child and not yet an adult. Many incisive changes take place in that period. Body growth, in a remarkable upward surge, attains its maximum for the individual. The physique assumes its characteristic configuration. Sexual development reaches procreative capacity. There is a striving after emancipation from sheltered existence, a trend toward increasing self-dependence in thought and action. The sphere of interest and participation expands from the confines of home, neighborhood and school to the community at large. The choice of a vocation, theretofore a playfully considered matter, becomes a real issue. Amorous stirrings seek a variety of outlets. Current standards and precepts are submitted to criticism not as yet leavened by the tests of experience. The established order is boldly challenged and then, after some struggle, gradually appropriated with more or less reservation.

A combination of innate soundness, wholesome childhood, and guidance from understanding elders helps most adolescents to feel their way safely through the groping and floundering which often precede maturing stabilization. Overreaching or falling short of the target can be corrected by bringing aimer, aim and target into more comfortable harmony. But the great

variety of backgrounds and personalities is fraught with possibilities of less easily smoothed conflicts and clashes. Inherent weaknesses and peculiarities, early exposure to educational blunders, and burdensome life situations may work singly or together to intensify perplexities and twist attitudes and performances. Hurdles taken by many with sufficient skill and economy of effort may come to loom as conspicuous barriers, circumvented inefficiently, avoided shrinkingly, or allowed to inflict serious damage in clumsy and incongruous attempts to surmount them.

Factors in the Development of Mental Instability.— The normal adolescent has his puzzlements and preoccupations; trouble starts when the puzzlements and preoccupations threaten to have the adolescent. It is then that the tasks of accepting one's own specific make-up, fitting oneself to things and people as they are, and establishing and maintaining give-and-take relationships come to a crucial test. Sensitiveness concerning somatic appearance and functioning may become a predominant feature connected with too much self-observation and logically as well as empirically inadmissible causal associations. Existing superstitions and pamphlet lore, sometimes echoed even by physicians, make of "self-abuse" a focus of worry and dread, especially perilous when a poorly resisted urge to masturbate, literal acceptance of ominous predictions, and ingrained ("constitutional") tendencies combine themselves to distort a young person's faith in his future.

Enslavement throughout the years of childhood by means of excessive parental pampering or domination creates fetters which the strong and sound can throw off with reasonable success; the weak and unstable often abandon themselves to continued passivity or make a hectic and occasionally wrecking affair of the proverbial adolescent rebellion against the older generation. The search for social, occupational, political, religious and cosmic orientation confronts the fledgling with ill-defined generalizations which he can brush aside without much ado, explore in pseudo-philosophic ruminations and debates, eventually sift for practical usage, or confuse to a degree out of proportion with utility and reality. Ambitions may fall in line with expectations and serve as guides for consistent planning, lag behind expectations through want of ability, purpose,

initiative or self-confidence, or shoot far beyond the attainable mark in daydreams but little related to actualities or in jerky, misdirected, prematurely spent thrusts.

Many young people, well prepared biologically and educationally, go through the second decade of life without essential disturbance. Some get temporarily entangled but emerge steadied, pulling themselves up on their own bootstraps or, if need be, holding on to a guiding hand. Others may become so thoroughly engulfed in all-embracing moods, ensnared in abstractions, trapped in blind alleys of mystic preoccupations, drawn away from realities, or rudely tossed into realities, that they are in serious danger of getting lost in their efforts to find themselves and their place in the scheme of things. These are the persons whose road to adult socialization often leads over bumpy detours, from which the main highway may or may not be regained. The detours, more or less profound deviations of feeling, thinking and acting, may take the shape of overpowering anxieties, obsessions and compulsions, hysteric reactions, delinquent behavior, alcohol and drug addiction, depressions and elations, and disorders of the schizophrenic variety.

Certain types of organic illness may in adolescence, as at any other age, disrupt the pattern of performance, hitting a hitherto healthy person, as in the instances of brain tumor, encephalitis or cerebral trauma, or bringing to the fore the consequences of congenital damage, as in the case of juvenile paresis. The feebleminded, whose early scholastic failures have been attenuated by play, physical strength and other compensations, may in adolescence present major problems to themselves and the community when they come to grips with vocational and social necessities and find the roads blocked.

"Mental disturbances" in adolescence are therefore manifold and require individual diagnostic and therapeutic consideration. This is best demonstrated by a number of examples. The following brief case presentations have been culled from the ward, dispensary and children's service material of the Henry Phipps Psychiatric Clinic of the Johns Hopkins Hospital. They have been chosen from among patients whose acute troubles were studied a sufficiently long time ago to make possible an evaluation of the outcome in the light of subsequent events.

CASE ILLUSTRATIONS

Case I. Rebellion of a Feebleminded Girl against Unattainable Expectations through Temper Tantrums, Stealing and Sexual Misconduct; Good Adjustment in Simple Environment.—Selma's poor progress in a small country school was for years interpreted as evidence of laziness. Her pharmacist father, a strong believer in "education," had proudly seen his older daughter emerge as a teacher and demanded impatiently that Selma do likewise. The family, mostly professional men and women, held those of less intelligence in the sort of contempt in which some wealthy people used to hold the poor; they had "divorced themselves" in shame of an uncle and cousin who earned their living as truck drivers. The father, whose declining health gave him presentiments of death, wanted quick results and sent Selma to an expensive boarding school, which dispatched glowing reports of her accomplishments.

When, at fourteen years, the girl still could barely do third grade work, she was taken to a physician, whose endocrine therapy did not bear out his promise to brighten her up. Meanwhile, she had begun menstruating, considered herself grown-up and met parental restrictions with sullenness and temper tantrums, chose her companions from the lower strata, tried to buy friendship from girls with gifts stolen from her father's drug store, from boys by making unmistakable sexual advances. Her mother took her to the psychiatric clinic, where she was found to have a mental age of below eight years, an intelligence quotient of 52.

Sterilization was recommended and carried out. Her father died shortly afterwards, leaving her enough insurance money to make it possible to board her on a farm with patient, understanding people. Relieved of scholastic pressure, at a distance from her sister and the neighbor girls with whom she had always been contrasted, she is happy, likes to help around, has given up her tantrums, gains recognition for her small services, and is generally well adjusted in her environment.

Case II. Prison Psychosis in an Imbecile Delinquent, Whose Proper Placement in a State Hospital Was Interfered with by a Psychiatrically Unintelligent Lawyer and Judge.—Leonard was the congenial member of a feebleminded family who lived in a shack in a Maryland county. He behaved satisfactorily at home and at school, where he was promoted "according to size," and passed through the seventh grade, never learning to read and write. When he was seventeen years old, the family moved to a nearby town, where his father got a W.P.A. job. In order to get money for movies, candy and knickknacks, Leonard broke at night into an office building and stole a typewriter and an oil stove. He was easily detected, taken to the court and sentenced to a short term of imprisonment. While in jail, he conducted himself peculiarly, complained that his food was poisoned, sang religious hymns, conversed loudly with God and angels.

Referred for psychiatric examination, Leonard was confused, spoke vaguely of enemies, was distracted by imaginary

voices. Upon the psychiatrist's recommendation, he was committed to a state institution. His parents, persuaded by neighbors to take him out, found a lawyer who filed a petition for a writ of habeas corpus, and a judge who released him. Within less than a month, he tried to steal in broad daylight from a store, had a quarrel with the proprietor who caught him in the act, and vindictively threw a stone through the store window. He was sentenced to a year in jail, where he now resides.

Case III. Schizophrenic Reaction with Passivity Feelings and Dramatic Ecstasies, Followed by Improvement and Resumption of Premedical Studies.—Richard, a quiet, seclusive, religious boy, became at seventeen years upset by his mother's illness with cancer and a premedical course which he took with the intention of becoming a psychiatrist. He was admitted to the clinic in November 1934, after he had wandered undressed from the college dormitory, spoken of hearing voices and being poisoned, and declared that he was a prophet. He had an ecstatic experience, in which he saw God as a cloud of dust and heard God talk to him, obeying what he thought to be God's commands. He identified himself with Christ. He was alternately restless and preoccupied, laughed inanely, demanded circumcision and operation for brain abscess. He expressed feelings of love for his mother, hatred of his father, guilt over intercourse with a girl years before. He built a coffin into which he placed himself symbolically by putting his glasses and a deck of cards within. A period of shallow self-accusations was followed by a burst of antics, animal-like performances, and grimaces.

Richard improved markedly after therapeutic induction of sterile meningitis and was discharged to an aunt's farm in July 1935. Three months later, upset by his mother's death, he experienced a recurrence of the illness and was readmitted to the clinic. Shock therapy brought slow but steady amelioration. He left the hospital in May, 1936, considerably improved, and in 1938 reported in a not too clearly composed letter that he attended college, finishing his premedical course.

Case IV. Schizophrenic Reaction in a Boy of Unstable Stock, with Abandon to Abstractions after Preoccupation with an Oratorical Contest; Complete Dilapidation.—Herbert, sensitive about the impression he made on people, had avoided companions since twelve years of age. At nearly fifteen, he won first place in an oratorical contest on the comparative merits of ancient and modern heroes. He disliked the ensuing publicity, became tense, hid in the cellar, read treatises on astronomy, cosmogony and psychology, filled a notebook with vague warnings and admonitions, mumbled to himself, spoke of death and world's end. His parents, crediting him with a "research mind," were not alarmed until he claimed that he heard bells and roaring engines when none were to be heard, touched all lamp posts along the street,

was careless in personal hygiene, ate salt and sugar by the handful, screamed for days, and kicked his mother.

Herbert had had several convulsions in infancy. He had a slight external strabismus. He had studied assiduously, participated in ball games, played chess and checkers, cultivated a garden. There were one suicide, some hospitalizations for mental illness, and several nonhospitalized social failures in the family. His mother kept her children in complete bondage, often with the aid of smothering demonstrations of affection, fainting spells and violent tantrums. She and her husband disagreed constantly and noisily. An older brother succeeded in emancipating himself after a terrific struggle which almost wrecked him.

When admitted in February, 1934, Herbert was preoccupied, inaccessible, spoke in monosyllables, jerked his head, maintained odd postures, giggled to himself, refused to eat, was severely constipated, tore up and chewed bits of paper. He spent two years in private hospitals, one year on a farm, is now in a state hospital, mute, occasionally grunting, sometimes smearing feces about the room, often masturbating—a total loss.

Case V. Temporary Withdrawal with Hypochondriacal Features as a Reaction to Transition from School Routine to Vocational Demands; Slow but Definite Recovery.—Julius, a quiet, shy, inconspicuous, studious boy, offered no particular problems until eighteen years of age. Following his brother's appendectomy in October, 1931, he had attacks of abdominal pain, vomiting, and fear that his appendix had burst. He graduated from high school with honors in February, 1932. Instead of looking for a job, he was listless, sat around the house, would not speak to anyone. Kink in the right ureter was diagnosed in August, 1932.

The boy's absorption with his physical condition caused his admission to the psychiatric clinic in September. He complained of many aches and pains, muscular twitchings, lack of strength and energy. He said, "My mother wanted me to get a job and I was scared. In school it was routine but when I got out it was a different world." He was confused about the divergent "diagnoses" made by the numerous physicians be had consulted. His notions about the functions of his body were corrected. He was reintroduced to daily routine procedures. He became interested in occupational therapy. Nephropexy was performed. He was discharged as improved in November.

Julius continued to keep to himself and remained idly at home, though his hypochondriasis had subsided. However, in 1934, he gradually went out more, took boxing lessons, and finally went to work. He has been working since then, is described as very ambitious, has a wife and a child, and is a good provider.

Case VI. Psychotic Episode Based on Sexual Malorientation; Good Recovery and Life Adjustment.—Robert, who in preschool age had lost his father through suicide, got on well at home and at school, where he progressed nicely and was active in athletics. At twelve years, he began to masturbate. Exposed to popular notions, he believed that his "secret vice" showed on his face, people looked knowingly at him, and he was not like others. He later assumed a similar attitude concerning nocturnal emissions. Connecting those with the food he ate, he tried to overcome them by eating very little and suspected people of trying to influence his ideas about eating. He lost weight, became constipated, had headaches, could not concentrate, slumped in his studies, thought that everybody watched him.

When admitted to the psychiatric clinic at sixteen years, Robert was undernourished, anemic, shy, diffident, blushed easily, declared he was "different" because of nightly self-abuse. For the first time, he had an opportunity to speak and get straightened out about his sex perplexities; his sources of bewilderment were taken up openly and directly. His interests broadened, his appetite returned, he regained his earlier ambitions. After his discharge, guided by the understanding family priest, he resumed his studies, took honors in an English university, taught high school. At last hearing, he was happily married and finishing his work for a Ph.D. degree.

Case VII. Suicidal Attempt of a Girl Failing in School and Wrongly Suspected of Sexual Misconduct.—Annette had "not given a day's trouble" until fourteen years of age, when school work became too difficult for her. Soon afterwards she underwent an appendectomy, following which she failed in her grade, refused to attend school, avoided her friends, ate little, slept poorly, was irritable, unhappy, "disgusted with herself." Two months later she was operated on for imperforate hymen. Her morbidly jealous father, who had often falsely accused his wife of infidelity, expressed suspicion that the operation had been the result of an aborted pregnancy. When, shortly thereafter, Annette came home later in the afternoon than expected, he said, "I have lost all my confidence in you," and threatened to send her to a reformatory. Annette went out, bought mercurochrome and witch hazel and drank the contents of both bottles. She was treated in a nearby hospital.

When seen in the psychiatric service, Annette was dejected, in utter despair, but capable of relating her story. At her own suggestion, she was sent to relatives where, in a friendly atmosphere, she gained distance from her domestic misery and scholastic hardships and recaptured her composure. When she came home a few months later, her father no longer questioned her decency and allowed a boy friend, of whom he approved, to visit Annette and take her to the movies occasionally. An-

nette again became sociable, friendly, reasonably happy, though not too fond of school.

Case VIII. Reactive Depression with Sex Preoccupations and Guilty Feelings in an Emotionally Unstable Adolescent; Life Adjustment Cannot be Clearly Foreseen.—Ernest was admitted to the clinic at nineteen years in a depressed condition in December, 1933. Aroused by "a terrible lusting feeling for a young girl," he had sought relief in masturbation, with subsequent revulsion and self-recrimination. He was preoccupied with the shape of the female body. His feeling of sinfulness was augmented by the perusal of religious tracts. He wished to secure salvation by confessing his sexual fancies. Not being able to recall all the women at whom he had "looked," he decided to make public his confessions in newspapers and on the radio. He spoke of having inherited his moodiness from Schiller, the German poet, whom he claimed as his great-grandfather.

Ernest was orphaned at an early age and was reared by relatives. He came of stable stock, except that an older brother was troublesome because of nonconformity, skirt chasing and "running wild" in general. He was sociable and popular; occasional emotional outbursts were ascribed to an artistic temperament. He was interested in drawing, painting and modeling. He delved into encyclopedias and emerged with an array of undigested facts which he liked to pour forth for the education of his companions. He progressed fairly well in a boarding school, where at thirteen years he was introduced by schoolmates to masturbation and homosexual practices.

At the clinic Ernest appeared dejected, full of self-reproaches, worried about "eternal damnation," expressed the need for atonement, found concentration and communication difficult, felt uneasy in the morning and more alert toward night, complained of insomnia, had a marked bradycardia (52 beats per minute). He responded readily to reassurance, showed increased interest in ward activities, formed friendships with other patients, gained several pounds, slept more and more soundly, and became more and more cheerful. He was discharged as markedly improved in March, 1934. He stayed for a year with a cousin and is now studying art in Chicago. He still expresses himself in rather vague and "borrowed" terms.

Case IX. Tics, Truancy, Irresponsibility, Childishness, Lying, Stealing, Carried over from Prepuberty and Overcome during Adolescence.— Alvin was brought to the children's psychiatric service in 1931 at ten years of age with the complaints that he was babyish, acted in a spoiled-child manner, bit his nails, blinked his eyes, shrugged his shoulders, was untruthful, supplied an assortment of aches and pains as a means of evading school attendance and home chores. He had started out as a much displayed child prodigy, had skipped grades, but then his ambition flagged and, despite an intelligence quotient of 134, he did very poorly in his studies.

He came of a home kept in constant turmoil by a domineering, driving, chronically excited, matrimonially disappointed mother, whose spineless husband had and claimed no voice in domestic affairs. He was the youngest of three children. His brother had, shortly before graduation from high school, become a traveling salesman in order to get away from home. His sister took refuge in a whining, complaining demeanor. The maternal grandfather, schizophrenic for many years, had died in a state hospital, and several aunts and cousins were very unstable.

The clinic could do little to decrease the mother's agitations. She resisted every attempt to remove Alvin from his home. The more she pushed, nagged, bribed and threatened him, the more he rebelled. He played truant, repeated grades, stole, lied, had numerous tics, masturbated frequently, reacted to maternal tantrums with tantrums of his own. But as he grew older, he felt more and more the need for greater freedom from his mother. He often came to the clinic on his own initiative to get help in trying to revise his conduct. He gradually dropped his stealing, story telling, truancy, shoulder shrugging, and temper tantrums, and he masturbated much less. He learned to accept his mother's excitements with forgiving humor as part of her personality.

To underscore his newly achieved self-reliance, Alvin left school while in the eleventh grade and got a job, continuing his studies in night school. From time to time his mother still came bursting upon the clinic with vague, unspecified complaints, with a glimmer of hope that he might be restored to her domination. She would rather have him back with all his earlier faults, at the time so distressing to her, than see him grow away from her grip. Alvin solved this problem by securing a position in a distant city as salesman for a publishing company. He is now, at nineteen, a sociable, well-liked, efficient, honest, responsible young man, of whose previous difficulties nothing remains save occasional mild blinking. When his business trips or a holiday take him to Baltimore, he sometimes reports to the clinic, which he considers as his emancipator.

COMMENT

Classification of Mental Disturbances.—Even the few examples cited illustrate vividly the great variety of behavior disturbances, etiologic factors, manifestations, therapeutic requirements and eventual developments during the interval between puberty and attainment of maturity. Reaction forms in puberty, more nearly than in childhood, are differentiated along the lines of adult psychopathologic patterns. Therefore, the same kind of general grouping is applicable:

Anergastic reactions—personality defects on the basis of irreversible cerebral tissue damage through infection (encephalitis, general paresis), neoplasm or trauma; behavior disturbances associated with the epilepsies or "cerebral dys-rhythmias."

Dysergastic reactions—transient, delirious, stuporous, comatose, confusional states on the basis of toxic, infectious, metabolic, circulatory illness.

Oligergastic reactions—various degrees of inborn intellectual deficits. (Cases of Selma and Leonard.)

Thymergastric reactions—sweeping mood disturbances in the form of depressions and elations, either congenital and often recurrent or alternating, or essentially situation-determined, "reactive." (Case of Ernest.)

Parergastic reactions, "schizophrenia," "dementia praecox"—bizarre distortions of feeling, thinking and performance with odd misinterpretations and mystic experiences. (Cases of Richard and Herbert.)

Merergastic reactions, minor psychoses, psychoneuroses—a vast variety of complaint disorders including neurasthenic, hysterical, hypochondriacal, obsessive-compulsive conditions, fear states, fainting spells, tics, and other substitutions for more effective and serviceable responses to life situations. (Cases of Julius, Robert, Annette and Alvin.)

Problems of Diagnosis in the Cases Presented.—The essential diagnostic requirements depend less on the allocation of a given problem to any of those reaction sets than on the sizing up of the salient features of the difficulty and the person who presents it. All of the boys and girls whose troubles have been sketched above have found themselves in one sort of a jam or another. Selma tried to help herself against scholastic pressure and unpopularity with temper tantrums, stealing and sexual aggressiveness. Leonard, who had gone on satisfactorily on the farm, came into conflict with the law when town life put unaccustomed temptations in his path. Annette attempted suicide when she could no longer cope with her father's innuendoes and failure in school after absence through sickness. Julius went into a hypochondriacal slump with anxiety attacks when he could not face the sudden shift from successful academic application to the demands of vocational initiative.

Robert and Ernest were both upset by masturbation and sexual fancies; Robert's parergastic-like emphasis was mainly on "the others," from whom he differed, who spied on him, influenced his ideas about eating; Ernest's thymergastic-like emphasis was on himself, his unworthiness, sinfulness, eternal damnation. Herbert, vulnerable heredo-biologically, mismanaged educationally, stirred to high pitch by an oratorical contest, got himself inextricably entangled in abstractions. Richard, thrown out of gear by serious illness of his mother to

whom he was enormously attached, blasted all relation to reality in abandon to ecstatic mysticism not bounded by recognition of natural possibilities. Alvin's rebellion against maternal domination spent itself for years in hypochondriacal and antisocial antics. Everyone of those adolescents responded to his own specific situational constellation in his own specific, unwholesome manner.

Many young people are confronted with scholastic demands not commensurate with their capacities or inclinations. Many young people are exposed to economic stress, physical handicaps, popular notions about masturbation, religious conflicts, vocational quandaries, educational errors of their parents. Yet the majority tread or fight their way through adolescence with reasonable efficiency and emerge with some kind of serviceable solution of their problems. They are helped by their ability to make use of their assets without being stumped by the obstacles; they are also helped by the inspiration from adults or even coeval friends. The boys and girls of our series lacked such directive and self-directing assistance, at least until they were turned over to the psychiatric clinic. They lacked the personal strength and vision necessary to handle perplexing situations and allowed the situations to overwhelm them.

Etiologic Factors.—Much has been said about the contribution of *heredity* to the development of psychotic conditions. Familial instability certainly plays its role but just as certainly is not the sole determinant and sometimes not even a part determinant. Herbert and Alvin were both descended from stock loaded with mental illness; yet, while Herbert fell by the wayside, Alvin succeeded in marching healthily onward on the road of life. Much has been said about the role of *environment*. Everyone of our cases indicates the importance of environmental factors. But each case also shows that situational impacts in themselves do not produce psychotic disturbances unless they hit susceptible people at the time of greatest vulnerability. Much has been said about the role of *complexes* and *mechanisms*. But the preoccupation with such speculative concepts and schematizations does not differ fundamentally from adolescent involvement in other kinds of abstractions. Much has been said about the connection between the *type of illness* and its *outcome*; "dementia praecox" was said

to terminate in irreparable disaster. Yet of the two patients who would be placed in this category, only Herbert went permanently out of circulation, while Richard made a creditable social recovery. No general slogan of heredity, environment, inferiority or any other complex, no oversimplifying reduction to terms of neurology or biochemistry, no correlation of prognosis with a "diagnostic" name picked from a limited nomenclature can possibly do full justice to the uniqueness of each patient and his troubles.

Principles of Treatment.—The therapeutic program is based on this uniqueness. It may require attention to *physical* disorders; nephropexy was a necessary step in the treatment of Julius, who had a kink in the ureter. It calls for knowledge of the patient's *intellectual status;* absence of such knowledge had exposed Selma to unwarranted expectations and caused a judge to enable Leonard to commit further delinquencies. It necessitates a full and expert study of each patient's *personality* and personal circumstances and all that is constructive within and without him. Sometimes, as in the case of Alvin, work with the patient himself offers the best opportunities. Often much can be gained with the help of parents, other relatives, physicians, teachers, clergymen, social workers, young men's and young women's clubs. Sometimes the patient can be adapted or readapted to an essentially unaltered environment; sometimes the environment must in varying degree be made to fit the patient's needs. Emotional conflicts, sexual bafflements, theological bewilderments and vocational confusions must be straightened out with and for the patient.

When things have gone wrong, the patient is best put into the hands of an experienced psychiatrist, without first resorting to all sorts of half-measures and slipshod fumbling. A human career is at stake. A few bottles of tonics or sedatives or a Y membership do not then suffice to regulate life's path for a ruffled adolescent.

But psychiatrists often do not get to see adolescents *before* and *unless* things have gone wrong. Most modern colleges, it is true, have their psychiatric advisers to whom students can be referred or turn spontaneously for guidance. Many high schools have vocational counselors who, familiar with the individual pupils' aptitudes and performances, can prevent a good

deal of occupational maladjustment. But on the whole the mental hygiene of the average adolescent is, and should by all means be, *the domain of the family physician*. Acquainted with most or all members of the family group, consulted about the children's health from the time when they are very young, he can better than anyone else get the feel of developments, relationships and directions, guide the establishment of wholesome daily routine, perceive the first little breaks, recognize and treat adequately hypochondriacal trends, help to overcome or sometimes even remove the source of somatic sensitiveness, reduce anxieties, be an understanding confidant in matters of sexual perplexity, and do many other things to keep the adolescent substantially fit. The family physician thus has a lion's share in the prophylaxis of mental disturbances of adolescents.

LIMITATIONS OF FOOD ALLERGY

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THERE is perhaps no branch of allergy that has stimulated more widespread discussion and interest than food allergy. During the past decade it has achieved a degree of importance in the medical and lay mind, perhaps not fully justified by the facts. New maladies have been added to the list of disorders in which "allergy" may be responsible, the possibilities of food allergy have been intensively studied and certain overenthusiastic early claims have been modified.

The term "food allergy" has sometimes been used to denote only that group of functional gastro-intestinal disorders which arise in certain sensitive individuals following the ingestion of a certain food, but now the term is more broadly applied to a larger group of functional disorders including asthma, allergic rhinitis, headache, gastro-intestinal disorders, urticaria, angio-neurotic edema, eczema and many other disorders believed sometimes to be due to the ingestion of certain foods to which the patient is sensitized.

Concomitant with the rapid development of food allergy there has arisen much confusion of thought relating to the fundamental nature of allergy. A brief review of the development of allergy will serve to re-define it and by comprehension of its definition one can gain a more accurate concept of the limitations of food allergy.

REVIEW OF THE DEVELOPMENT OF ALLERGY

Anaphylaxis in Animals.—The phenomena of anaphylaxis in animals were recognized by Flexner, Theobald Smith and Richet in the nineties. Richet was led to investigate the nature of anaphylaxis because it had been observed that guinea

pigs inoculated with a mixture of toxin and antitoxin (horse serum) for the standardization of diphtheria antitoxin often died following subsequent injection of horse serum, and because some patients receiving diphtheria antitoxin developed strange complications, later known as *serum sickness.* Further studies by Arthus, Rosenau and Anderson, and Otto led to a crystallization of our knowledge about anaphylaxis and subsequent studies have not materially altered the earlier concepts of the phenomenon.

Anaphylaxis may be defined as a state of unusual reactivity of certain organs or tissues to the injection of a foreign protein (toxic or nontoxic) following a primary sensitizing dose of the same protein. Even under identical conditions the reaction does not always occur. An incubation period of from three to ten days is necessary to enable the primary injection to sensitize the "shock organ" which will react when the second dose of the same protein is injected. The amount of the second dose may be the same or smaller than the primary dose. There is no transmission of the sensitivity to the offspring.

The Concept of Allergy.—Bostock first described hay fever as a clinical entity and years later it was conclusively proved by Blackley (1873) that it was due to exposure to pollen of certain plants. In 1905 Weichert, and in 1906 Wolff-Eisner suggested it might be an anaphylactic disorder. In 1910 Auer and Lewis described the outstanding anaphylactic reaction in the guinea pig as broncho-spasm and in the same year Meltzer suggested that bronchial asthma might be an anaphylactic disorder.

In 1911 Noon reported the first successful treatment of hay fever by the injection of gradually increasing doses of an extract of the offending substance, and in 1912 Schloss first reported the use of the skin test to discover foods which might be the cause of functional gastro-intestinal disorders and of eczema in infants and children.

Certain fundamental differences between anaphylaxis and the known facts relating to hay fever and asthma of the seasonal variety led to the universal adoption of the term "allergy," originally suggested by von Pirquet in 1906, to denote the new branch of medicine in which certain transient disorders occurred in a relatively small proportion of people following

inhalation or ingestion of, or contact with certain proteins and other substances to which the whole population is exposed. Although there are apparent differences between allergy and anaphylaxis, it is contended by some that they are fundamentally the same phenomenon.

The *hereditary tendency* toward transmission of the capacity to become sensitized was early recognized, for in no other way was it possible to explain why a small proportion of the population becomes allergic when the whole population is equally exposed to allergens. Having inherited the capacity to become allergic, the individual may develop one or more allergic disorders, but he does not necessarily develop the same ones his ancestors had.

Cooke's Postulates.—Robert A. Cooke in 1922 formulated a series of postulates to be fulfilled before a substance may qualify as an allergic etiologic agent: (1) Specific sensitization must be demonstrated by (*a*) a positive local reaction, cutaneous or ophthalmic, or (*b*) the original allergic manifestation must be artificially reproduced at will on introduction of the substance either inhaled, ingested or subcutaneously injected. (2) It must be shown that the individual has come into contact in some way with the suspected substance in order to permit it to act as an etiologic factor.

Thus, it may be seen that Cooke has defined certain conditions under which a substance may be considered to be responsible for producing an allergic symptom complex, just as Koch's postulates define conditions under which a certain bacteria may be considered responsible for producing a given infectious disease. Many allergists have accepted Cooke's postulates as valid under most circumstances, but with the mushroom growth of food allergy some investigators have been led to largely ignore them.

THE ERA OF FOOD ALLERGY

Although it was early recognized that foods played an important role in atopic eczema in children, and in certain cases of gastro-intestinal disorder, urticaria and angioneurotic edema in both children and adults, food allergy was believed to be relatively uncommon when compared with the incidence of inhalant allergy.

In 1927 Vaughan directed attention to the allergic nature
of migraine. In a group of thirty-three patients having this
malady he found twelve whose migraine was thought to be
allergic in origin. These patients gave positive skin tests to
certain foods; by eliminating these foods from the diet, the
patients could be kept free of headache, and by reinstating
them, headache could be reproduced. Thus, Vaughan's twelve
cases fulfilled the classical requirements necessary to qualify
foods as allergens in twelve cases of migraine.

The spectacular results reported by Vaughan in this com-
munication precipitated a wave of interest and enthusiasm
in food allergy with rather far-reaching consequences. Further
studies of migraine and of common forms of headache and
extension of interest to the domain of the functional gastro-
intestinal disorders eventually led some investigators to the
conclusion that Cooke's postulates could largely be ignored in
food allergy, although their essential validity was recognized
in inhalant allergy. Their clinical studies of these patients led
them to conclude, for example, that a certain food may pro-
duce symptoms *only once* after eating it ten or fifteen times,
thus violating the very essence of the definition of an allergen
as described in Cooke's postulate (1, *b*) in which it is stated
that, "the original allergic manifestation must be artificially
reproduced at will on introduction of the substance either in-
haled, ingested or subcutaneously injected."

The assumption that a certain food may be allergenic only
once in a while led to the conclusion that skin testing is highly
unreliable, for food testing under this circumstance generally
yields a negative skin test. To account for the frequent occur-
rence of symptoms it is usually necessary to incriminate a
considerable number of foods. Sometimes attacks are seem-
ingly precipitated by certain foods often enough to preclude
the possibility that their ingestion is purely coincidental in
relation to the onset of an attack, but even so, many allergists
are inclined to believe the fundamental etiologic factor is not
allergy. Under such circumstances there is perhaps no more
reason to believe allergy is the prime etiologic factor than
there is to regard drafts as the primary cause of the common
cold. Foods may in some way precipitate attacks at times
but the mechanism is not understood.

PRESENT CONCEPTS OF FOOD ALLERGY

The main body of allergists at present incline toward adherence to the strict definition of an allergen as expressed in Cooke's postulates, with the exception that there is no compelling reason to believe a positive skin or ophthalmic reaction is a *sine qua non* of an allergen. Thus, it is believed that if a certain food is allergenic for a certain individual it should be possible to reproduce his symptoms in a high percentage of trials, by feeding larger than average portions. In the highly sensitive patient, attacks are precipitated regularly with small portions of food—indeed, only a trace of some foods is necessary to produce an attack.

In the case of some foods, *heat* destroys the allergenic factor. Some patients can tolerate evaporated milk but not raw milk. A hardboiled egg is sometimes tolerated in egg-sensitivity. Ratner and Gruehl, in a study of the anaphylactogenic properties of certain cereal foods and breadstuffs, conclude that certain cereal grain products subjected to moist heat, undergo physicochemical changes characterized by the coagulation and flocculation of soluble protein fractions, thus reducing their antigenicity. Well cooked meat is sometimes nonallergenic, while ingestion of meat cooked "medium" or "rare" will cause symptoms.

The *speed of absorption* of food allergens has been amply studied by Walzer and his associates. In 1927 Walzer reported a simple direct method of studying the absorption of undigested protein in human beings. Using the passive transfer technic (Prausnitz-Küstner) he took blood from a patient highly sensitive to peanut and injected the serum into the skin of a nonallergic individual, thus sensitizing the skin to peanut at the site of injection. Following the ingestion of peanut the nonallergic person developed itching, redness and wheal formation at the sensitized spot, thus marking the entrance of the peanut antigen into the systemic circulation. Employing this technic Gray and Walzer found an average absorption time of 24.3 minutes after oral administration, 18.6 minutes after feeding by duodenal tube, and 18.7 minutes following rectal administration. However, when an ordinary meal composed of several foods is eaten, it is reasonable to assume that a longer time may elapse between the meal and the occurrence

of symptoms.   The allergic reaction generally takes place within a few minutes to several hours following food. Walzer has shown that the longer absorption of food from the intestine is delayed, the less likely it is to produce symptoms, presumably because the digestive processes have destroyed to a large extent the allergenic factor in the food.

Symptoms occurring late following ingestion of a specific food have been reported; these patients give a negative skin test, and the reaction occurring twenty-four to forty-eight hours later is believed due to sensitivity to some decomposition product rather than to the original food.

Unaltered food antigen absorbed from the gastro-intestinal tract is believed to be responsible for symptoms in food allergy. Gray and Walzer have demonstrated absorption of peanut antigen from the rectum where there are no digestive enzymes. Furthermore, absorption of food antigen from the vagina and uterine cervix, from the intact skin and from the peritoneal, pericardial and pleural cavities, has been demonstrated, thus showing that food antigen may be absorbed independent of the action of digestive enzymes.

DIAGNOSIS IN FOOD ALLERGY

History.—The history should be recorded with great care. First, a general anamnestic survey is made so that all possible organic diseases may be ruled out by appropriate diagnostic procedures.   Then, inquiries are directed toward discovering possible external factors such as inhalants, foods and contactants which might be responsible for symptoms.   In making inquiries about foods, a detailed knowledge of the *composition* of foods is essential, for a patient may report that he has eaten no wheat products yet he may have eaten gravy not knowing that it contained wheat flour.   Numerous other examples of unsuspected ingredients in common foods could be cited.   A list of foods the patient particularly likes may reveal foods eaten in large quantities; and a list of foods he dislikes may give a clue to possible allergens, for sometimes he instinctively avoids allergenic foods.   A history of earlier allergic disorders in the patient and a family history of allergic disorders give additional support to the possibility that his present illness is allergic in origin.

Skin Tests.—Skin testing is done by the scratch technic or the intradermal technic. The latter is 100 times more sensitive than the former, hence many allergists employ both tests, for if the patient is highly sensitive one is less likely to produce a constitutional reaction if he uses the weaker scratch test first. All foods giving a negative reaction are then retested, employing the more sensitive intradermal test.

In the *interpretation of skin tests* there are differences in opinion as to what constitutes a positive test. Some allergists regard only wheal formation with a surrounding area of erythema as a positive test while, on the other hand, some regard as suspiciously positive small areas of ·redness and similar reactions occurring twenty-four to forty-eight hours after testing.

False positive tests occur frequently and their significance can be determined only by the method of clinical trial wherein one attempts to get relief from symptoms by eliminating all foods giving a reaction or suspicious reaction, followed by reinstatement of these foods one at a time to determine which foods produce symptoms. Rackemann believes false positive tests indicate past sensitivity to these foods.

False negative tests have been widely discussed. Some believe them to be rare, while others believe they occur commonly. False negative tests are generally ascribed to differences in the freshness of foods and to alteration of the allergenic substance due to cooking. Food testing extracts are always made from raw fresh foods. Malkin and Markow, in a comparative study of intradermal tests using solutions prepared from raw and from cooked foods, made 652 pairs of tests; they found the same reaction in 91 per cent; in 3.9 per cent the raw extract reacted positively against a negative cooked test and in 4.3 per cent the cooked extract reacted positively against a negative raw test.

Leukopenic Index.—The leukopenic index was introduced by Vaughan as a possible means of discovering allergenic foods. Behind it is the theory that foods to which the patient is sensitive will cause a leukopenia following ingestion. It has not been proved that the test is reliable, only one food can be tested in a day, and the technic is tedious and time-consuming. In 1939 Vaughan states: "We must conclude that the

leukopenic index is still in the experimental stage and cannot be discussed at this time as a routine diagnostic procedure in allergy."

Food Diary.—The food diary often reveals possible allergenic foods. The diary must be kept long enough to include at least several attacks. So that all possible ingredients of foods are known the patient is limited to foods prepared at home. A study of the diary will show what foods may be eaten without producing symptoms, and from the list of foods eaten twenty-four hours before the attack it may be possible to discover one or more that has not been taken during the time when he was symptom-free. The possible allergenicity of these foods is then determined by the method of clinical trial and by skin testing.

Elimination Diets.—In those patients believed to be allergic to foods, and in whom other means of discovering the allergenic foods have failed, the four elimination diets devised by Rowe are helpful. If the patient becomes symptom-free on one of the four diets, one food at a time can be added until the allergenic foods have been discovered. If the patient does not become symptom-free on one of the four diets, it is highly unlikely that food allergy is responsible for his symptoms.

COMMON MALADIES IN WHICH FOOD ALLERGY MAY OCCUR

Certain maladies coming on in sudden attacks which gradually subside leaving no demonstrable pathologic changes have become known as allergic disorders, although nearly all may in many instances have a nonallergic etiology. In any of these disorders in which allergy is the cause, foods may prove to be the allergen; however, in some, inhalants are believed largely responsible.

Perennial Asthma.—The nonseasonal type of asthma occurs from time to time throughout the year, although some cases may have seasonal exacerbations due to sensitivity to a pollen. The allergens generally responsible are the inhalants, such as animal dander, feathers, dusts, spores and chemicals; sometimes inhaled foods such as wheat flour cause asthma. Asthma may be caused by ingested foods also, but the incidence of asthma due to foods is likely not high.

In a review of 1074 cases of asthma, Rackemann found

only eight in which foods were proved responsible for attacks; six were sensitive to only one food; one was sensitive to two foods and one was sensitive to a variety of foods but he does not state whether all caused asthma. However, in children with asthma he believes foods are more commonly responsible, and in his experience eggs, wheat and cow's milk are the chief offending foods.

On the other hand, some observers report a substantially higher incidence of food allergy among asthmatics. Tuft believes some are overlooked by placing too much reliance upon skin testing. In discussing the variations in the reported incidence of food allergy he states, "Such wide disagreement is no doubt due to the variation in the method of testing for food sensitivity (scratch or intracutaneous) and also partly to the fact that negative skin reactions are often obtained even in the presence of definite clinical sensitivity."

Allergic Rhinitis (*Vasomotor Rhinitis, Perennial Hay Fever*).—The swollen, boggy pale nasal mucous membrane associated with a watery nasal discharge, marked nasal obstruction, sneezing and itching is the same clinical syndrome seen in hay fever of the seasonal type due to pollens. In cases of long standing the nasal mucous membrane often becomes hypertrophied and polypoid growths may appear. Secondary infection of the mucous membrane, with involvement of the sinuses, is a frequent complication.

As in asthma, the inhalants are the most frequent cause. Again, there are wide differences of opinion relating to the etiologic role of food allergens. Many observers report that they not infrequently find foods responsible for this malady. In sharp disagreement, however, is the analysis of 198 cases observed over a period of one to ten years, reported by Winkenwerder and Gay, in which they found not one case due to foods.

Urticaria and Angioneurotic Edema.—These are considered together because the underlying pathologic processes are essentially the same. Urticaria involves chiefly the corium, while angioneurotic edema involves the corium and subcutaneous tissue.

Urticaria may appear in the form of ordinary hives or it may be the size of a hand, a large erythematous area surround-

ing a central wheal. Urticaria tends to occur in crops coming suddenly and leaving gradually, only to be followed by another crop.

Angioneurotic edema is a painless, circumscribed swelling which appears rather suddenly and disappears gradually. Distribution of the lesions is asymmetrical and the sites of predilection are the eyelid, lips and tongue, but they may occur anywhere.

The reported etiologic factors in urticaria are many and varied; perhaps in the majority of cases an allergic factor cannot be demonstrated. In those cases in which an allergic origin can be proved, foods and drugs are the main offenders. Most of the reports of satisfactory treatment by elimination of foods are subject to criticism, in that the patient was not followed long enough to actually determine the outcome. In a review of 170 cases of urticaria and angioneurotic edema followed for a period of from two to ten years, Fink and Gay find 30 per cent due to focal infections (allergic to bacteria), 20 per cent due to allergy and the remaining 50 per cent due to other causes. The chief factors causing symptoms in the allergic group were the following foods: eggs, wheat, meat, strawberries, oranges, apricots, celery, potato, tomato, peas and nuts; drug sensitivity, however, was responsible in a few cases in this group.

Atopic Eczema.—The term "eczema" has been applied loosely to designate a great variety of skin disorders. From this group there have been extracted the allergic eczemas, a group with well defined clinical characteristics and a specific etiology. The allergic eczemas have been divided into two groups, according to the location of the lesion and the mode of sensitization. First, there is the contact dermatitis in which the lesion is in the epidermis and occurs in the parts exposed. It is due to exposure of the skin to external influences such as chemicals, oils of plants, various dusts and a great variety of other substances. Secondly, there is the atopic eczema (atopic dermatitis; neurodermatitis) in which the lesion occurs in the corium, has a fairly typical distribution and has one or more specific blood-borne etiologic agents.

Atopic eczema starts with itching followed by scratching which eventually results in papulelike formations and thicken-

ing of the skin. It has a tendency to locate in certain areas, notably the antecubital and popliteal fossae, the back of the neck, the face, the inner and upper surfaces of the thighs, anterior and external surfaces of the legs, the external surface of the forearm, the palms and soles and the genitalia. It may be limited to one or two areas but sometimes it involves most of the body.

In atopic eczema foods are most frequently the *exciting agent*; environmental factors such as dust, feathers, animal danders, orris root and silk are believed in some cases to be either the primary cause or a complicating factor. In infants and young children, the responsible food can sometimes be recognized by obtaining a history of onset of eczema shortly after the addition of a new food to the diet.

Elimination of foods which give positive skin tests is often followed by rapid disappearance of the eczema in infants and young children. Older children and adults usually do not respond as readily as do some in the early age group, likely due to the development of lichenification. The experiences of dermatologists have led them to expect improvement in only a limited number of cases following elimination of foods.

Headache.—Some cases of migraine and of common forms of headache are believed to be allergic in origin.

Migraine may be described as a severe type of headache, often unilateral but sometimes generalized, associated with nausea often leading to vomiting, and with transient visual disturbances at the onset. Disorders of vision, however, are not a constant feature of migraine. The attacks occur at irregular periods in most cases but in some migraine appears about the time of menstruation.

In the general course of events, the patient subject to migraine may have an attack only once in two, three or six months' time, and not infrequently the period between attacks is longer. During periods of emotional stress or of pressure of activity, associated with fatigue and insomnia, the patient disposed to migraine may have attacks lasting one to three days with an interval of only one or two days between attacks.

The fulminating nature of the migraine attack in association with nausea and vomiting followed presently by recovery suggests a similarity to the anaphylactic reaction. The ten-

dency toward hereditary transmission of the malady has been amply demonstrated. The observations of Goltman in his patient with migraine who was subjected to exploratory craniotomy, indicate an area of angioneurotic edema at the site of the craniotomy during attacks of headache. Vaughan, Rowe, Balyeat and others have presented classical evidence that food allergy is the cause in some cases of migraine.

It would seem there is ample evidence that some cases of migraine have an allergic origin, yet many clinicians having broad experience with migraine contend that they have never encountered an authentic case due to allergy.

Those who are not impressed by the role of allergy in migraine believe they offer a more direct approach to the patient's immediate problem, which is frequently recurring attacks, by studying the source of the patient's *nervous and emotional instability* and helping him to make such adjustments as are necessary to restore him to vigorous health. Rest and freedom from responsibility alone often result in spectacular improvement. This form of treatment has much to commend it, for the allergic approach is long and tedious owing to frequently changing food sensitivities. Furthermore, once the patient is free of his frequently recurring attacks, he often loses interest in those which occur at long intervals. Where it is possible to find foods which more or less consistently cause migraine, the allergic approach to treatment is undoubtedly the method of choice.

Some cases of *simple headache* (those not due to organic disease) have been proved to be due to food allergy but what has been said regarding treatment in migraine, is equally applicable in this form of headache.

Gastro-intestinal Allergy.—There is no clearly defined syndrome which may be regarded as characteristic of gastro-intestinal allergy. The symptoms are essentially those of the acute abdominal conditions, hence great care must be exercised in eliminating every possibility of organic disease requiring surgical intervention before considering the possibility of an allergic origin.

Nausea and vomiting, diarrhea and acute abdominal crises with urticaria, angioneurotic edema and purpura are sometimes due to food allergy. The acute abdominal condition in

some cases resembles appendicitis or an "acute gallbladder." The attacks come on within a minute to several hours following ingestion of food and a great variety of foods may be responsible. Diagnosis of an allergic disorder is sometimes made at the operating table when the surgeon finds localized areas of edematous bowel instead of one of the usual acute abdominal conditions. In children cyclic vomiting and pyloro-spasm are sometimes due to food allergy.

Chronic Gastro-intestinal Disorders.—These deserve special consideration. The presenting symptoms may be one or more of a variety of complaints referable to the gastro-intestinal tract. Commonly one encounters feelings of discomfort, of heaviness or of fulness in the abdomen, which may be general localized or may shift from place to place. Frequently the patient complains of abdominal pain, which when described more accurately is said to resemble a heavy weight, needles and pins, burning and other paresthesias. While describing these symptoms the patient's facial expression indicates much greater distress than he is able to express in words. Other symptoms include belching, flatulence, constipation, diarrhea, sometimes with passage of mucus, heart burn, regurgitation of food, nausea, loss of appetite and partial or complete vomiting of a meal. These symptoms in various combinations have been described as "indigestion," "dyspepsia," "chronic appendix," "chronic gallbladder," "ulcer" and "colitis."

The patient frequently volunteers the information that certain foods disagree with him and often these have been avoided but without improvement; finally it would seem to him that all foods must disagree for he is unable to find foods which will permit freedom from symptoms.

Upon direct questioning one may learn that the patient has many symptoms characteristic of the *affective* (*emotional*) *disorders* or of the *psychoneuroses*. Although more distressing than the digestive symptoms they are often not revealed for several reasons: (1) the patient believes they are secondary to his digestive disorder; (2) he is reluctant to discuss them because full discussion often leads to highly personal matters; and (3) the symptoms are difficult to describe in words.

A great variety of mental and nervous symptoms may be described. Mental depression or melancholia, worry, impair-

ment of the power of concentration, loss of interest, anxiety, fears and insomnia are often the most distressing symptoms. Feelings of tenseness, the sensation of a band around the head, numbness and many other sensations may occur.

The only objective evidence of abnormality is *spasticity of the colon* as revealed in a routine series of gastro-intestinal x-rays.

More than 200 patients of this type have been under my observation in hospitals or nursing homes for periods of time ranging from eight or ten weeks to six months. I have had ample time to study the patients in every detail and have always kept in mind the possibility of co-existent food allergy, but I have yet to find one whose digestive symptoms could be attributed to allergy by either a strict or a reasonably broad interpretation of the definition of an allergen.

The reported incidence of gastro-intestinal allergy and of toxic mental states due to food allergy would undoubtedly drop if more careful consideration were given to the psychiatric aspects of these cases coupled with an appreciation of the fact that spontaneous remissions and exacerbations of symptoms are common and that many run a self-limited course with recovery.

Conceding the possibility that low-grade food sensitivity may co-exist in some cases, they are, however, essentially problems in the borderline between psychiatry and medicine. The psychiatrist sees those whose mental symptoms are severe and the others are divided among the gastro-enterologist, the allergist, the internist, the endocrinologist and the chiropractor.

SUMMARY

A tendency toward a broad interpretation of the definition of allergy, accompanied by reports of cases inadequately studied and not followed for a sufficient period of time, has led to undue emphasis upon the prevalence of food allergy. The main body of allergists have adhered to the strict definition of allergy, with the result that their work in many instances does not confirm the observations of their confreres.

A review of the common conditions in which food allergy may occur has been presented. Wide discrepancies in the reported incidence of food allergy in these conditions have

been observed. Most likely, future studies under more uniform conditions as to method of diagnosis will present a more accurate account of the true incidence of food allergy in these maladies.

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THE MANAGEMENT OF PULMONARY ABSCESSES

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Etiology.—The majority of lung abscesses can be ac-
counted for on the basis of inoculation of atelactatic or, less
often, infarcted lung tissue by organisms from the upper air
passages. The atelectasis does not need to be massive or even
lobar, but may involve a much smaller area; this lung tissue
then having lost much of its resistance to infection may become
secondarily infected by aspiration of pathogenic organisms
from the nose and throat. Atelectasis may occur during the
course of any type of pneumonia; it frequently follows opera-
tions, especially those in which deep and prolonged anesthesia
is employed; it occasionally results from aspiration of foreign
body or of vomitus. Aspiration of material from the mouth
into the lower bronchi takes place during operation under any
type of anesthesia and even during normal sleep. A much
smaller proportion of lung abscesses are embolic in origin,
occurring after operations or during puerperal or other pelvic
infections.

Bacteriology.—The bacteriology is far from uniform and
depends to some extent on the duration of the abscess and the
etiologic factors involved. For example, in cases that follow
pneumonia the *pneumococcus* or at times the *streptococcus*
may predominate, at least during the acute stage. Later,
however, as the abscess becomes more chronic the flora changes
and usually conforms rather closely to that found about the
teeth in cases of pyorrhea. In addition to this we have a situ-
ation in which the bacteria in the infected area have to become
accustomed to multiplying in a walled-off abscess in which

partial anaerobiosis is maintained. As a result of this the flora in chronic abscesses often becomes predominantly an *anaerobic* one, the bacteria most frequently encountered being anaerobic or facultatively anaerobic streptococci and bacilli. Various *aerobic organisms* are often found in association with these; namely, alpha hemolytic (viridans) streptococci, staphylococci, beta hemolytic streptococci and others.

Other mouth organisms which are frequently reflected in the flora of the pulmonary lesion are *fusiform bacilli* and *spirochetes* which, in the majority of cases, are found in only small numbers and appear to be secondary invaders. However, occasionally fusospirochetes are present in such overwhelming numbers and the lesions respond so dramatically when intravenous arsenicals are given that they seem definitely to have been the ones primarily responsible for the abscess.

In the case of embolic abscesses the bacteriology at first is less complex and consists mainly of one type of organism, such as the *hemolytic streptococcus, colon bacillus, staphylococcus* and again particularly the *anaerobic streptococcus*, which is so often found in puerperal and pelvic infections of all sorts. However, later the abscess becomes secondarily contaminated by the mouth organisms, and the flora then becomes very similar to that found in the other chronic abscesses.

Prophylaxis.—This brief consideration of etiology and bacteriology therefore leads us naturally to the first and most important step in treatment, namely that of *prevention*. This consists in the following measures:

1. Strict daily care of the teeth and gums, especially before any operations, particularly those upon the upper respiratory tract.

2. Avoidance in all operations of an irritating, deep and prolonged anesthesia which is likely to result in excessive production of mucus and abolition of the cough reflex long enough to allow aspiration into the smaller bronchi.

3. The position of the patient during operation should be one to favor drainage toward the mouth whenever possible, rather than into the lower bronchial tree.

4. Sedatives ought not to be given in large doses, for this results in depression of the cough reflex, shallow breathing and stasis of secretions.

5. All secretions developing in the large bronchi or trachea during the period of anesthesia should be aspirated with nasal catheter suction.

6. As the patient awakes there is danger of aspiration of vomitus which can be prevented by elevating the foot of the bed or by further use of suction.

7. Finally, postoperative inhalation of CO_2 and O_2 in order to hyperventilate the lungs is an invaluable method of preventing stagnation of mucus and other material that may lead to atelectasis. This is best carried out on the operating table and every few hours thereafter for two or three days.

Differential Diagnosis.—The majority of cases of pulmonary abscess offer several interesting possibilities in diagnosis, which is frequently arrived at mainly by exclusion. Of course in the typical case in which the abscess follows an operation, we have a *history* of cough and fever usually within the first few days, then often pain in the chest and a few days later expectoration of a large amount of sputum having a foul odor. *Physical signs* are apt to be minimal, but the finding of a few localized rales possibly accompanied by bronchovesicular or tubular breath sounds are important localizing signs. The pulmonary signs vary a great deal from day to day; at times they may be absent entirely and the next day they may present signs of cavitation and consolidation. The *x-ray shadow* is at first rather nondescript and ill-defined, but later becomes more sharply demarcated and usually at some time in the course of the infection will show signs of a cavity or cavities with fluid level. *Clubbing of the fingers* is a variable finding but when present is very helpful in the diagnosis; it sometimes develops within two weeks of the onset of the abscess. In such a typical case or in that which follows an attack of pneumonia the diagnosis of pulmonary abscess is fairly certain, but in a good percentage of the cases the onset is an insidious one, and it is then necessary to exclude several other conditions before the diagnosis is justified.

In *pulmonary tuberculosis* the x-ray shadow is at times very much like that of a pyogenic infection, though it is usually more diffuse and splotchy in character. Repeated concentrated sputum examinations, at times obtained from the stomach washings, at times by bronchoscopic aspiration, special cultures

for tubercle bacilli and inoculation of guinea pigs, will yield positive findings in the majority of cases of tuberculosis.

Carcinoma of the lung and bronchus is more common in the age group above forty years and a common early symptom is cough with repeated small hemoptyses. However, because secondary infection of the lung in the region of the tumor is a frequent occurrence, the symptoms and signs are often those associated with lung abscess.

The x-ray shadow is usually more dense toward the hilum, with radiation out into the lung parenchyma. Bronchoscopic examination is of prime importance in establishing the diagnosis of bronchogenic carcinoma; by this method the new growth can often be seen and biopsied; at other times the bronchus can be seen to be compressed by an extrabronchial mass. Other important findings indicating the likelihood of neoplasm are atelectasis of a lobe or the occurrence of a bloody pleural effusion. Sometimes the diagnosis is not made until operation on the supposed lung abscess, at which time the unusually firm wall of the abscess may be noted and a section of it taken for histologic examination. Another useful measure is the lipiodol bronchogram which may show obstruction of one of the bronchi; while this is often found also in uncomplicated abscesses, it is a more common finding in patients with lung tumors. Metastatic lesions must always be looked for, the most common sites being the supraclavicular glands, the bones, the liver and the brain.

Bronchiectasis is usually, though not always, limited to the lower lobes, and the history of profuse expectoration is apt to be one of several years duration. The plain x-ray may show no more than an increase in the root shadows extending into the bases, but the x-ray after lipiodol instillation shows definite dilatation of the bronchi. These dilatations are sometimes cylindrical, at other times fusiform or saccular with occasional large bronchiectatic abscesses. It is often impossible to tell whether or not a chronic lung abscess has had its origin in a bronchiectatic cavity.

Encapsulated empyema or *interlobar empyema* is often responsible for fever, an area of decreased pulmonary resonance, suppressed breath sounds and clubbed fingers; moreover, the x-ray picture is apt to be very much like that in

pulmonary abscess. In this condition, however, the patient seldom has a profuse expectoration of purulent sputum unless the empyema has broken into a bronchus. Localization is carried out by x-rays taken in various positions, and, following this, exploratory aspiration may be attempted if one is reasonably sure that the lesion is extrapulmonary, and if he is careful not to enter the lung itself.

Other less common conditions to be considered are *fungus infections* of the lung; *subphrenic* or *liver abscess* extending up through the diaphragm; *mediastinal* or *pulmonary infection* from perforation of the esophagus, usually due to carcinoma; *Hodgkin's disease* with not only mediastinal glandular enlargement but occasionally with involvement of the lung itself; *aneurysm of the aorta,* etc.

MEDICAL MANAGEMENT

Once the diagnosis of pulmonary abscess has been established, a program of therapy is outlined and adhered to rigidly; in this way conservative treatment may be given a thorough trial before resorting to surgery. It is at times both gratifying and surprising to see large areas of pulmonary consolidation with cavitation clear up completely in this way, when it seemed almost certain that surgical drainage would be necessary. However, surgery should not be delayed too long, for once the abscess has become a chronic one the outlook following any type of therapy becomes increasingly bad. We feel that, in the great majority of cases, if there has not been definite improvement both by symptoms and by x-ray after three to five weeks of medical management, the problem then becomes a surgical one.

General Measures.—The regimen of medical treatment is one that aims at building up the patient's resistance, combating the infection, and attempting to secure drainage through the bronchi. This means, first of all, *rest* in bed, a *high caloric, high vitamin diet,* plenty of *fresh air* and a *high fluid intake.* *Steam inhalations* containing *creosote* and *benzoin* are often helpful, especially when the cough is severe and unproductive, in liquefying the secretions and in soothing the bronchial mucosa. Creosote also has the effect of rendering the odor of

the breath and the sputum less offensive. *Potassium iodide* or *ammonium chloride,* by mouth, can likewise often be used to advantage for making the secretions of the bronchi less viscid. It is, of course, important not to suppress the cough too much, and consequently *cough mixtures* containing codeine or other narcotics are to be avoided except for the purpose of quieting the cough enough to allow the patient to rest.

Arsenicals.—Intravenous arsenicals, usually in the form of *neoarsphenamine,* are often ordered routinely in cases of lung abscess, but most authors are of the opinion that arsenicals are worthless unless numerous spirochetes and fusiform bacilli are found in the sputum. Even in these cases, though the breath and sputum usually become less foul, the course of the abscess itself is often little affected. However, there is a small percentage of pulmonary infections in which these organisms are found in great abundance in the sputum. Such abscesses are usually of short duration and not well localized, and often clear up rapidly under treatment with these drugs, in such a way as to make it appear that the Vincent's organisms were the ones primarily responsible.

The percentage of these cases of pulmonary fusospirochetal infections is quite low in the series of cases of lung abscess reported by most authors, but others have found a rather high proportion of cases coming under this category. We have observed a small number which seemed to derive marked and rapid benefit from intravenous neoarsphenamine, the usual dosage being 0.3 to 0.6 gm. every three or four days for approximately three weeks. Used as a routine measure, however, it has proved disappointing. Various preparations of *bismuth* may be given intramuscularly in conjunction with the neoarsphenamine.

Other Drugs.—An additional drug which certainly should prove valuable in the rare cases due to beta hemolytic streptococci is *sulfanilamide.* In the cases in which the abscess directly follows pneumococcal pneumonia and in which the suppuration is presumably due, at least in part, to the pneumococcus, sulfapyridine or *sulfathiazole* should be tried early. At the present time the latter drug seems to have an equally satisfactory effect against the pneumococcus and a more marked one against some of the other bacterial organisms asso-

ciated with pulmonary abscesses. Consequently, it would seem best to give sulfathiazole a thorough trial in early cases until one is sure whether or not there will be any benefit from it. However, it is questionable whether any of these drugs will have any effect on the lung abscess once it is fully developed, as walled-off abscesses have never yet been found to respond favorably to chemotherapy; nor have any of this group of drugs been found effectual against *anaerobic bacteria*, and inasmuch as these organisms apparently play the most important role in the chronic abscesses, it is not likely that they will add much to the therapy except in rare cases.

Iron in various preparations is useful in combating the anemia that so often is present.

Vitamin preparations should be used for those patients who show any signs of vitamin deficiency, or who are not able to take a diet containing adequate amounts of vitamins. Moreover, vitamin stores of the body are apt to be depleted during the course of any chronic disease or infection, this being particularly definite in the case of vitamin C, an excess of which should always be present in the diet of patients with pulmonary infections. There is considerable experimental evidence indicating the importance of adequate amounts of vitamin A in the diet in maintaining the integrity of the epithelial lining of various structures, including the bronchi. Lack of this vitamin may lower the resistance of such tissues to bacterial invasion, therefore it is especially important to guarantee an abundance of this factor by supplementing a diet already high in vitamin A with daily doses of one of the fish liver oils.

Postural Drainage.—If drainage of the abscess can be accomplished through the bronchus, every effort should be bent toward promoting this result, before considering surgical intervention. Many patients can be started at once on the road to recovery by means of postural drainage. Knowing the position of the abscess and the direction of the bronchi draining it, the proper posture for the most adequate drainage can, as a rule, be easily determined. However, the use of this posture should not be persisted in if the drainage is not good, for a patient often finds by experimentation which position produces the best results.

The simplest method is to have the patient lie over the side

of, or across the bed with his hands on the floor or on a low chair and with the trunk turned so that the affected side of the lung is uppermost. In many cases various positions will have to be tried. The patient should be instructed not to cough hard at any time, but to allow drainage to take place mainly by gravity, with just enough cough to help keep the passages clear. A patient is often wrongly advised to cough vigorously during . this period of drainage; in addition to the obvious dangers to the old or debilitated, there is the risk of spreading the pulmonary infection, for during a hard cough secretions in the smaller bronchi may be forced backward into the bronchioles and alveoli, rather than outward toward the trachea. This is the probable cause of the high fever and pneumonic spread of the infection that is often seen in patients with lung abscess or bronchiectasis who are receiving vigorous postural drainage. Consequently, when the drainage by this means is not satisfactory, or when the patient cannot refrain from severe paroxysms of coughing, it should be discontinued.

The scheme ordinarily followed is to have the postural drainage carried out for five minutes twice a day, gradually increasing it to ten or fifteen minutes two or three times a day. However, this intermittent type of drainage is certainly far from ideal, for during the rest of the day and especially at night when the cough reflex is depressed there is cessation of drainage, and possible spread of the infection to other parts of the lungs following the course of gravity.

Localization of the abscess should be carried out as precisely as possible by means of x-rays, bronchoscopy and careful physical examination, and a plan of postural drainage outlined accordingly. The drainage should be as nearly a continuous procedure as can be arranged without too much discomfort to the patient. Some employ *special beds* for this continuous drainage, beds which can be tilted at various angles depending on the location of the abscess. The same results can be obtained for abscess in the *lower lobes* by raising the foot of the bed gradually until the patient is accustomed to having it elevated about 20 inches, at the same time the involved side can be kept uppermost the greater part of the time. In the case of *upper lobe* abscesses the best plan is to have the patient in Fowler's position during most of the day and night,

the head and trunk are then lowered at intervals during the day in order to allow easier drainage of the purulent secretions from the larger bronchi. Postural drainage being a procedure of such importance in the therapy of pulmonary abscess, its technic should be carefully explained to the patient, who should be supervised until he understands and masters the methods.

Pneumothorax.—This form of therapy has been given extensive trial, but the results of its use have been very unsatisfactory. There is always the danger of producing an empyema, especially in the cases in which the abscess is peripherally located, and in addition to this danger the pneumothorax seldom leads to collapse of the thick-walled abscess, or may even cause kinking of the bronchi thus increasing the difficulty of drainage through the bronchi. The only type of lesion in which this procedure is justified is that which is located close to the hilum. In such cases the risk of inducing empyema is much smaller and there is a chance of aiding drainage by the air's exerting a more uniform pressure on the walls of the abscess.

Bronchoscopic Drainage.—Routine bronchoscopy is a very important step for location of the abscess, for detecting carcinoma when possible, and for locating and removing foreign bodies. Many times it may promote better bronchial drainage by clearing viscid secretions from the bronchi and by shrinking swollen mucous membranes, thus opening the passages. Likewise, by this means it is possible to remove, by cauterization, granulation tissues that are obstructing the bronchus. Repeated drainage by this method is rarely necessary, however, since equally good results can usually be obtained by postural drainage, with much less discomfort. It certainly should be tried, particularly in the early acute abscesses. If drainage is improved and the patient tolerates the procedure well and shows general improvement, it may be well to repeat it. However, long periods of bronchoscopic drainage are very seldom indicated.

Transfusion.—This is helpful in many cases and should not be withheld until the patient is very anemic from loss of blood. Used early, even if there is only slight anemia, this is an additional procedure in the medical regimen that often seems to be of marked benefit. It is also a necessary step in

most cases in preparation for surgery or immediately after operation.

<h2 style="text-align:center">SURGICAL TREATMENT*</h2>

It is impossible to formulate any definite rules as to when surgical measures are indicated. Each case is a problem unto itself and one must be guided by the course of the infection in the individual case. Conservative treatment should certainly first receive a painstaking trial and be continued as long as there is definite improvement both by x-ray and in the general condition of the patient. Operation should not be delayed long because the chances of ultimate recovery then become rapidly worse. The arbitrary time limits of medical treatment which seem to us adequate in the majority of cases are from three to five weeks. If, at the end of that time, improvement is not definite, surgery should then be resorted to. The abscesses which are located peripherally usually do not drain as well through the bronchus and so are less apt to clear up under medical treatment; in them, surgery is usually indicated earlier than in the more centrally situated ones in which the medical regimen is more apt to prove effective and surgery more hazardous.

The most satisfactory surgical treatment in most everyone's hands has proved to be *external drainage*, done preferably in two stages. Safe surgical drainage requires accurate localization of the abscess, and the determination of its nearest point to the parietal pleura. Guides to this localization are (1) the position of pain when present; (2) physical signs and (3) x-rays taken in various positions. There is often a temptation to put a needle into the chest in order to confirm the localization of the abscess before operation; this is a dangerous procedure, owing mainly to the hazard of spreading this virulent infection throughout the pleural cavity, and is definitely contraindicated for this reason.

The first stage of the operation consists in rib resection with exposure of a wide surface of pleura. The wound is then packed for at least forty-eight hours. At the end of this time, using the actual cautery, the abscess cavity is opened widely

* The surgical treatment is that recommended by Doctor George G. Finney of the Johns Hopkins Surgical Staff.

and the cavity packed with gauze. There are a few cases in which the parietal and visceral pleurae are adherent when first exposed, and it is then possible to carry out the drainage in one stage. However, if there is the slightest question as to whether the pleural surfaces are adherent, the two-stage operation is utilized.

Lobectomy and *pneumonectomy* have been carried out successfully by a number of surgeons, but these procedures are only applicable to a few selected cases. At the present time it seems that the only cases in which these operations are indicated are chronic ones of many years standing in which most of the functioning lung tissue of one side has been destroyed by infection, with widespread fibrosis and cavitation.

Postoperative Complications.—The most serious complications following operation are:

1. The spilling-over or squeezing-out of infective material from the diseased tissue into the larger air passages, at times causing complete *bronchial obstruction* with asphyxia. If this discharge of pus is not profuse enough to cause complete obstruction there is, nevertheless, the danger of spread of the purulent secretion into other parts of the lung, causing a dissemination of the infection. These dangers can be greatly lessened by clearing the bronchi preoperatively as completely as possible by postural or bronchoscopic drainage, and by utilizing the most gentle operative procedures.

2. *Pyopneumothorax*—usually a very virulent anaerobic infection, which makes the patient desperately ill, and the prognosis grave. This complication necessitates immediate operation, preferably some form of closed pleural drainage. Pleural irrigations with zinc peroxide solutions have been tried in a few cases with encouraging results.

3. *Hemorrhage*—occurring most often after operation on chronic abscess where the thick fibrotic walls do not collapse after drainage of the abscess. It is less apt to occur if the cautery is employed at operation; but when it occurs it calls for repeated cauterization, packing and transfusions.

4. *Persistent bronchial fistula*, which may need secondary operations for closure. This complication, though not a common one, is very troublesome and is most apt to follow drainage of long-standing chronic abscesses.

RESULTS OF TREATMENT

The results of the various forms of therapy for pulmonary abscess have been far from encouraging, the mortality in most series varying between 30 and 50 per cent. In some recent reports, however, the results have been much more encouraging, owing apparently to early operation. This is not a condition in which one can separate the medical from the surgical cures and say that one method is better than the other, because most of the surgical cases have been treated conservatively first and the outcome in every case is the result of this combined therapy.

The following conditions are those which impress one as offering the *chief barriers to a successful outcome* in these pulmonary infections: (1) chronic abscess—that is one whose duration has been over six months; (2) massive hemoptyses or postoperative hemorrhages; (3) patients in the older age groups; (4) alcoholism; malnutrition from any cause; chronic diseases such as epilepsy, in which aspiration may occur during a seizure; (5) operations performed in any part of the body in the presence of marked oral sepsis.

In addition, there is a type of case in which the infection runs a rapid and fulminating course from a few days to a few months, resisting all forms of treatment. Such cases show acute necrotizing lesions throughout the lung tissue and are often classified as *pulmonary gangrene.* These infections are probably due to massive inoculations with highly virulent material, of lung tissue whose resistance has been lowered in some way; the only therapy that seems to offer much hope is supportive, with transfusions, etc., until the process becomes localized.

SUMMARY

From a consideration of the various factors involved in the problem of pulmonary abscess it seems perfectly clear that the incidence and mortality in this condition can be markedly lowered.

Various methods of prevention have been discussed, which should help lower the incidence, particularly of the post-operative group.

Once the abscess has developed the patient's chance of cure depends mainly on early diagnosis and the institution of a care-

fully planned program of conservative therapy. In this medical program the particular importance is emphasized of a high vitamin intake, transfusion when indicated, efficient postural drainage, occasional bronchoscopic treatment when drainage is not satisfactory, and intravenous arsenicals in certain cases. By these methods we can expect a cure in about two thirds of the early cases and in a smaller proportion of the chronic ones; in the meantime, we should be able during this period of treatment, to improve the general condition of the patient enough so that if operation be necessary he will represent a much better surgical risk than if operated on during the first few days after admission.

Operation is indicated if a period of medical therapy of three to five weeks has proved unsuccessful. The more conservative type of operation, that is the two-stage procedure with rib resection and cautery incision of the abscess, seems to offer the best chance of cure.
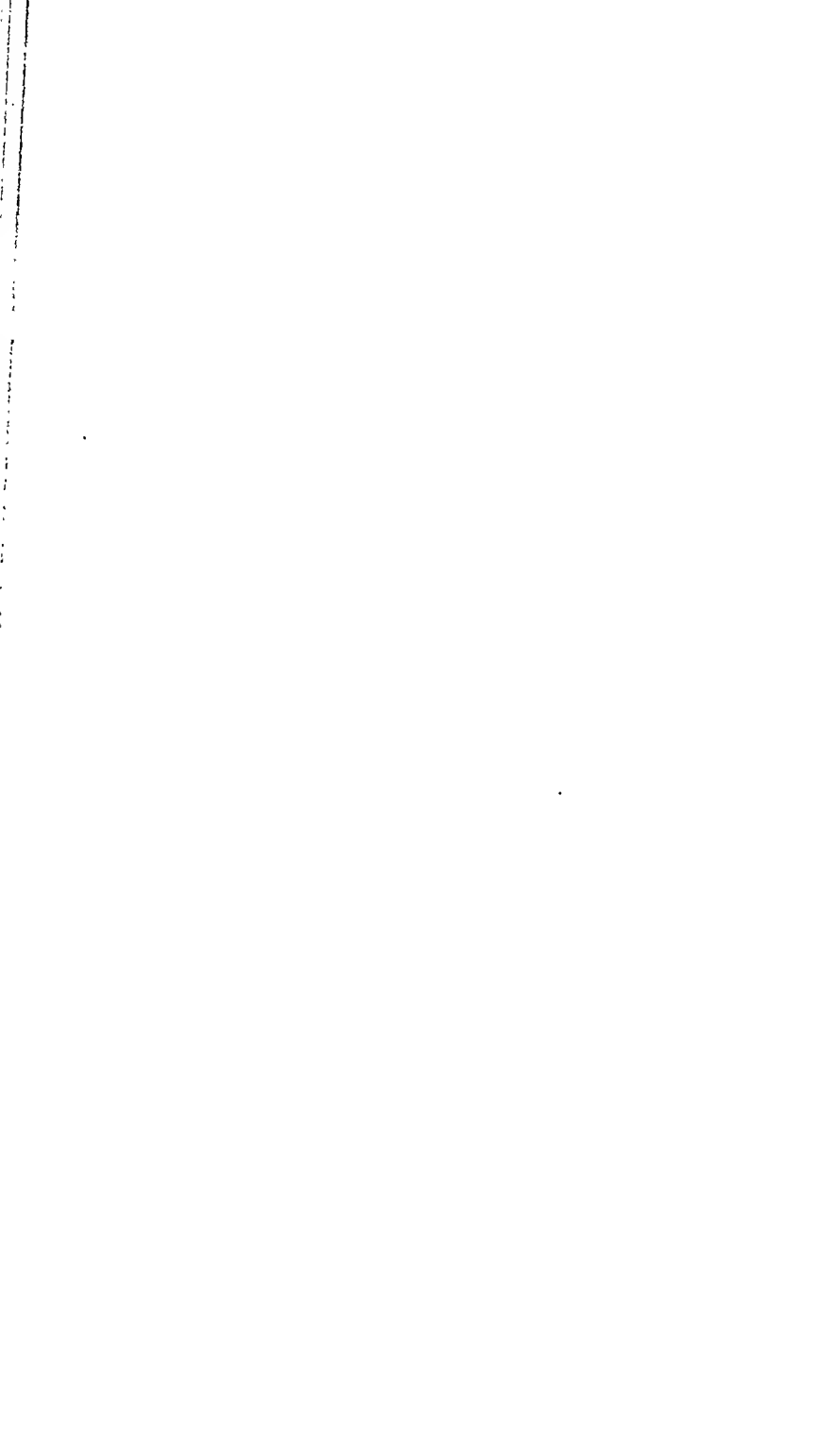
THE TECHNIC OF THE GASTRIC EXAMINATION

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THE technic of gastric examinations varies from the "Ask for a G-I series on Mrs. Smith, doctor; she is complaining of some vague dyspepsia" to that of the tyro who, lest something be missed, insists on doing everything "including the kitchen stove" to every patient who has a gastric complaint. Indeed, with the increasing number of laboratory procedures one is so easily inclined to lean more and more on them for the formulation of his diagnosis that he asks for more tests when those already done bring him negative reports. Dependence on the laboratory may make the diagnosis but it increases the labor of the overworked staff there and, because there is increased work, the cost to the patient mounts to unreasonable levels. Then, too, the formation of a clinical diagnosis is always so much more interesting than the ordering of laboratory studies!

THE CLINICAL EXAMINATION

The History.—History-taking I put first in the technic of the gastric examination and I justify its position with the aphorism whispered by a Continental gastro-enterologist of an older generation: "Listen to the patient, doctor; he is telling you the diagnosis!" Certainly there is no procedure in the gastric examination so important as the compilation of the patient's story of his trouble. It may be a tedious task, interpolated with much irrelevant material, but it usually supplies a key to the solution of the problem as well as the presentation of that problem. The humblest practitioner can take as excellent a history and get as much from it as can the university consultant. The patient may be a poor observer and often

the greatest patience on the part of the doctor is necessary to get at the facts.

From a good history of a gastric complaint one can often tell whether or not it is likely that the patient has organic disease and, if he has organic disease, whether it is in the stomach or whether the gastric symptoms merely reflect organic trouble that is located elsewhere. Perhaps there is neither organic disease nor reflex gastric symptoms from disease elsewhere, the gastric symptoms being entirely on a functional or nervous basis. We usually allow the patient to tell his own story in his own way and, of course, he tells his presenting complaint first. We try to list the symptoms as pain, gas, heartburn, etc., and to analyze them as to when they began, their location, whether or not they have come in attacks and, if so, what time of day the attacks began, their relationship to food intake, their duration, the means of relief, and usually we ask for the patient's own opinion as to the cause of the symptoms. In patients with gastric complaints it is often helpful to get the story of an average day when the complaints are present. This gives us knowledge of the influence of work, of rest, of food, of medicine, of posture, of bowel movements, of vomiting and so forth.

The patient with a stomach complaint is a person as well as a stomach and in the family history, in the past medical history, and in the social history we learn of that person and we can often obtain a very good idea of his intelligence, of his ability to withstand discomforts, and of the material of which he is made. We do not believe that the lists of questions with "yes-and-no" answers give the valuable diagnostic picture that a chronologic "story-history" gives. Often we check and re-enforce or contradict the history by a bit of observation in the hospital while the patient is on the ordinary American dietary and without treatment. Often one learns by such observation that leading questions were too leading to a poor observer and that the true story wasn't really like the history first obtained.

The Physical Examination.—Again I say that the patient is a person and not only a stomach; thus in the physical examination, as in history-taking, the entire body-machine should be looked over in the patient with gastric complaints. Of course at this clinic session when we will talk only of the

gastric examination we are particularly interested in the abdomen and more especially in the upper abdomen. Our extreme specialization at this moment reminds me of the story of a young chemist from this university who once visited a well known Continental university. When asked what he would like to see and to do, he replied, "I'd like very much to meet Professor So-And-So in your Department of Chemistry." The meeting was arranged and the Professor opened with, "So-o-o-o! You are another victim of your stars, a chemist!" "Well," said the Baltimore lad, "I'm interested in organic chemistry and particularly in the chemistry of dyes." "A-a-a-ah! That's my forte, too!" said the European. "But the azo dyes are really my field– especially the blues!" For us today it's the stomach only!

Abdominal examination usually reveals very little in diseases of the stomach, yet the simple signs of gastric disease must always be looked for because, when positive, they are extremely important. One must not miss the woods for the trees and become more interested in an achlorhydria than in an epigastric mass. The body contours and abdominal contours, particularly the costal angle, often give leads about gastroptosis and atony. The gastric silhouette with visible peristalsis may be seen, or a gastric mass may be noted on *inspection* alone. The old trick of distending the stomach with gas by giving tartaric acid and soda and so noting its size and position is historically interesting but quite unnecessary in these days of roentgenography. Relative flatness of the navel and evidences of collateral circulation are most suspicious signs of ascites.

Palpation will often reveal a gastric mass, a metastasis, tenderness, ascitic fluid, or hernia. Certainly, it is the most important physical measure in gastric diagnosis outside of x-ray. A succussion splash (clapotage) in the fasting stomach is most suggestive of pyloric obstruction, and, if present long after the last meal, it suggests poor gastric tone.

Percussion of the stomach was attempted by older clinicians to indicate its position but such percussion went out of fashion as the roentgenologic examination came in; in gastric disease it reveals very little except dulness in ascites.

In gastric disease *auscultation* is of little practical value.

THE LABORATORY EXAMINATION

It is by a multiplicity of laboratory examinations that the consulting gastro-enterologist impresses his patients and it is by the judicious selection of these tests that the busy practitioner saves time and money and confirms his clinical diagnosis. Indeed, it is a rather good rule to run only those tests which will confirm the clinical diagnosis, unless unlimited laboratory facilities are available or the gastric problem is an obscure one. I have no set routine in the laboratory.

The Fecal Examination.—One of the simplest and most important tests in gastric disease is the examination of the stool. This need not be particularly unpleasant and the simplest tests require only a moment. We have the patient bring fecal specimens after he has been on a meat-free, broth-free and meat-gravy-free diet for four days. These are sent to the laboratory in glass jars with screw tops or in half-pint, heavy, paraffin-coated containers which have tight lids. The color and formation of the stool is noted, as is the presence of mucus and gross blood. A microscopic examination is made for pus, parasites, and ova—but these have nothing to do with the gastric examination.

The Benzidine Test for Occult Blood.—Most important of all measures is the benzidine test for occult blood. A bit of stool the size of a pea is smeared on an ordinary glass microscopic slide that is held over a white background. On this is poured a solution of benzidine base in glacial acetic acid (this is made up every day or two). Then a few drops of hydrogen peroxide are added and the resulting color formation or the absence of change is noted for about thirty seconds. If the green or blue comes out immediately and is very intense, it is called a four-plus reaction; if it is very strong but is delayed a few seconds in its appearance, it is called three-plus; if it is of moderate intensity and comes out rather slowly, it is a two-plus reaction; still weaker reactions are termed one-plus, trace, and slight trace. The negative test and those reported to show only slight traces of occult blood are the most significant results of the examination because, by their delicacy, they quite definitely rule out significant ulceration along the gastro-intestinal tube, including the stomach. Positive tests are significant only if bleeding gums, nosebleeds, and hemorrhoidal ooz-

ing can be ruled out. This last means of "contamination" may be excluded by obtaining a bit of stool for the test through the sigmoidoscope from above the hemorrhoidal area. Whenever a strongly positive benzidine test is found persistently in the fecal specimens of patients with gastric complaints, an effort should be made to find the source of the bleeding.

We rarely use the *guaiac test* for occult blood in the stool; it is too often negative in the presence of slight ulceration which we know to be serious ulceration.

The Gastric Content.—The usefulness of the examination of the stomach content in the clinical diagnosis of disease of the stomach has been greatly exaggerated by some. I believe that it is rarely essential in the diagnosis of gastric disease but I occasionally do find it helpful, I confess. It's a real comfort to find a high gastric acidity in the case of an elderly person with pyloric obstruction, but acidity and achlorhydria may also give one a "bum steer" if the findings aren't interpreted in the light of the entire clinical picture. Most gastric disturbances are on the basis of motor dysfunction rather than secretory dysfunction.

The majority of patients whom I see have a natural distaste for any kind of stomach tube, and one has difficulty in believing that they are descended from the race of Americans that developed the fad of self-lavage of the stomach directly after the turn of the Century. Yet gastric gavage at the hands of their doctors is expected by most patients with chronic stomach complaints and, though it rarely yields information of great importance, it is an extremely simple procedure that may be of some value. Its chief value is to tell us whether or not the stomach can secrete acid—and then it is often wrong.

Stimulation of the Gastric Secretion.—Achlorhydria to an Ewald test-meal may not be an achlorhydria to histamine stimulation, and a stomach which shows an achlorhydria at the first histamine injection may secrete acid at a subsequent histamine test. There are various test meals of bread and tea, Uneeda Biscuit and water, Triscuit and water, 7 per cent alcohol, etc.

By us *histamine* is given subcutaneously in doses of 0.005 mg. per kilogram of body weight rather than in doses of 0.01 mg. per kilogram as is classical. Experience has made us feel

that the smaller stimulus is adequate and gives practically none of the unpleasant reactions of the larger doses. We prepare the histamine for injection by dissolving a 3-mg. tablet of Burroughs & Wellcome's Ergamine Acid Phosphate in 1.0 cc. of boiled water in a teaspoon. This tablet contains 1.0 mg. of histamine base so of course the 1.0-cc. solution contains that amount of histamine. With a tuberculin syringe the required amount of the drug is given. A man of 60 kilograms would be given 60 x 0.005 or 0.3 cc. of the solution.

Extraction of the Gastric Contents.—There are two methods of extracting the gastric content after a stimulus to gastric secretion: the single aspiration and the fractional aspiration. Since I do not believe the information obtained by fractional gastric analysis justifies the time and labor expended, we use the former method routinely. We ask the patient to take a slice of unbuttered bread or toast and a glass of water on a fasting stomach and to present himself to us from forty-five minutes to an hour later. With the old-fashioned Ewald-Boas tube we extract from 5 to 20 cc. of the gastric content for examination. We have no quarrel with those who wish to use smaller tubes of any type by the oral or nasal routes. The larger tubes are ever so much faster and so we use them. Rarely does the procedure take longer than twenty seconds (for which patients are universally grateful).

The patient sits in a straight-backed chair, takes out any removable dental work which he may wear, has a light cloth apron draped around his neck, inclines his head and body slightly forward, and is instructed to breathe through his mouth during the procedure. The tube is introduced into the pharynx and one or two swallows are asked for. With a series of rapid movements the tube is pushed forward to the mark, the aspirator is attached, and then the tube is rapidly withdrawn. No attempt is made to empty the stomach entirely. Indeed, the extraction is made so quickly that many patients hold their breaths during the entire procedure, though that isn't particularly to be desired. Usually most of the sample of stomach content is in the tube and it is allowed to run into a container.

Examination of the Collected Sample.—Simple titrations of the juice with N/10 sodium hydroxide against Topfer's reagent and phenolphthalein are recommended. In these days

of almost universal access to roentgenologic examination it is rarely necessary to test the gastric content for the organic acids or to look at it microscopically. The presence of occult blood in the gastric content (or in samples of feces) after gastric gavage means nothing, for the passage of all tubes introduces a traumatic factor. Motor meals are no longer needed because roentgenologic examination reveals all and more than they can tell us. Estimations of the amount of gastric secretion are quite unnecessary in the usual gastric examination. For research purposes such estimations are useful.

The *x-Ray.*—*Indications For Its Use.*—The territory of roentgenology has been invaded by the predatory specialties. The dentist, the orthopedic surgeon, the urologist, the thoracic diagnostician, and the gastro-enterologist have dismembered the roentgen empire and taken what they wanted. As the skiagrams of the chest have become almost indispensable in the diagnosis of thoracic disease, so roentgenologic examination must be accorded first place in the diagnosis of cancer, ulcer, diverticulum, and herniations of the stomach. The gastro-enterologist is likely to make at least a fluoroscopic examination of all the malfunctioning stomachs that come to him. He does this, first, because the history may point to intragastric (or duodenal) disease; secondly, because he wants to be thorough and miss no obscure trouble; thirdly, because the patient expects such an examination at his hands; and, lastly, because the study is relatively easy. On the other hand, the general practitioner rightly reserves x-ray studies of the stomach for those cases of hemorrhage from the upper gastro-intestinal tract, for the chronic dyspepsias, for elderly persons with relatively short histories of "stomach trouble," for patients with obscure anemias and persistently positive benzidine tests on the stools, and for those patients who present a syndrome of a well recognized gastric (or duodenal) disease. In older persons with vague dyspepsias of short duration, roentgen examinations of the stomach are being made more and more frequently and promptly and in this way more and more early carcinomata are being picked up.

Training and Experience Are Essential.—Though the roentgenologic examination of the stomach is simple in principle, training and experience with the method are essential. There

is a wide variation in the relief patterns and in the silhouettes
of normal stomachs; on the one hand is the transverse, steer-
horn stomach of the obese man with its suspicious-looking fun-
nel-like antrum; on the other hand is the atonic, ptosed fish-
hood stomach of the undernourished multipara. Normal
findings must be recognized and the commonest mistake in the
roentgen diagnosis of gastric disease is the diagnosis of path-
ology that is not present. We are often asked whether or not
roentgenoscopic examination alone is satisfactory to us. It is
usually satisfactory in separating normal from abnormal stom-
achs. However, in persons with direct fluoroscopic evidence
of gastric disease or in very obese patients we always ask that
films of the stomach be made. In insurance problems we al-
ways have films taken for record. Everything considered,
fluoroscopic examination of the stomach and a few films are a
little more satisfactory than either alone.

Technic of the Examination.—Our roentgenoscopic technic
is very simple. The patient presents himself in a fasting state
(or, if we have to so arrange it, directly after the extraction
of a sample of his gastric content). He stands before an up-
right fluoroscope and he is given in his left hand a 12-ounce
glass of a suspension of 4 ounces of barium sulfate in water
to which a little malted milk has been added. After a brief
screen examination of the abdomen for distended loops of
intestine and for opaque substances, such as bismuth, and after
a short examination of the chest, the patient is asked to turn
"left oblique." In this position he takes two or three swallows
of barium. The barium is followed down the esophagus and,
as it enters the stomach, the observer often obtains some in-
formation about the cardiac end. With this small amount of
barium in the stomach, the patient is turned to his original
position and the rugal folds are studied by palpation. Special
attention is paid to the posterior wall where, in our experience,
lesions are most often missed. Then the remainder of the
barium is drunk and the entire gastric silhouette is studied in
the upright, prone and supine positions. In this last position
we look carefully for small thoracic stomachs, our present
hobby. In patients who present stubborn pylorospasm we have
found that a ten-minute rest on a cot in an adjoining room does
more to relax the spasm than do the usual drugs. Five or six

hours after the ingestion of the barium a screen examination is made for delay in gastric emptying.

Interpretation of the Findings.—The *primary* or direct roentgenologic signs of organic gastric (or duodenal) disease are defects in the contours, mottled areas in the silhouette, and changes in the rugal pattern. *Changes in the contours* may be niches, an hourglass deformity, or any irregularity in the borders of the silhouette. In the stomach without food residue *mottling* may be produced by a polypoid tumor which displaces the barium or, rarely, by multiple ulcerations (Fig.

Fig. 52.—Spot film showing multiple ulcerations.

52). The *rugae* may be altered in size, they may converge on an ulcer, or they may be ironed-out in a localized area by a tumor.

Errors are made rather commonly by interpreting spasm from extragastric disease as an intragastric lesion and retained food (or food taken by mistake before the examination) may produce a mottling suggestive of a tumor. Coarse rugae on the greater curvature may produce a suspicious irregularity there and, on the other hand, such irregularity may represent a small neoplasm; here experience may make a wise roentgenologist prefer to describe the skiagrams and leave the diag-

nosis to the clinician, who has other data in the case—perhaps the report of a gastroscopic examination. Deformities of the stomach from external pressure are sometimes a source of error in radioscopic and radiographic diagnosis.

Secondary or indirect roentgenologic signs of gastric disease are the size and tone of the stomach, its rate of emptying, and some inflexibility of the gastric walls. These only tend to confirm a diagnosis; they do not make it.

The *bete noir* of the roentgenologist is the diagnosis of disease (or of the absence of disease) in the stomach to which the surgeons have done their bit. Obstructions may be demonstrated by roentgen study and sometimes an ulcer niche may be observed. However, mucosal folds are tricky and often the findings are incorrectly interpreted and often lesions are missed. We use the same technic in the study of postoperative stomachs as in the examination of the presurgical stomachs except that in the former group we spend a little more time in the study with small amounts of barium.

Though it may have pitfalls for the inexperienced, the *x*-ray gives us objective evidences of trouble in most cases of organic gastric disease. It is probably helpful enough to justify the kind of gastric examination which was requested by the visiting physician when he said, "Ask for a G-I series on Mrs. Smith, doctor; she is complaining of some vague dyspepsia." That visiting man with his method will not learn everything about his patient's stomach but he will learn considerable —and it will be with a minimum of effort on his part.

The Gastroscope.—In the past four years we have found ourselves using the gastroscope more and more in the study of gastric complaints. The Wolf-Schindler flexible gastroscope is an instrument which one may safely use if the contraindications to its passage are strictly observed.[1] We were slow in accepting any gastroscope because we felt that all of them were dangerous instruments and that only a limited area of the stomach could be seen with them. However, once one looks into the stomach through the gastroscope he exclaims, as we did, "How beautifully one can see!" And straightway he becomes a convert to the method. With the gastroscope we can see about four fifths of the interior of the stomach. The posterior wall of the upper portion is unfortunately a "blind area"

and sometimes the lesser curvature of the antrum isn't well seen, though, if there is peristalsis in the antrum, the mucosa of this second "blind area" will be seen to "unroll" before the examiner. The difficulty in gastroscopy is not the passage of the instrument; it is the interpretation of what one sees.

The Technic of Gastroscopy.—The technic of gastroscopy is not difficult. It is an out-patient procedure and hospitalization has not yet been necessary for any of our patients because of the examination. The patient, who has first been examined clinically and studied fluoroscopically so that we are certain that the esophagus is clear and that there is no systemic contraindication to the procedure, presents himself in a fasting state to the out-patient nurse an hour before the procedure is scheduled. The nurse, who is a great asset because of her kindly and reassuring manner, gives the patient 64 mg. of codeine and 0.4 mg. of atropine hypodermically on his arrival. He is allowed to lie down if he wishes until the time of the examination.

Before examination any removable dentures are taken out and the pharynx is *anesthetized* with 4 or 5 cc. of pontocaine, which is sprayed through a Schindler anesthetization tube. Five minutes later this topical application is repeated. We formerly used a 10 per cent solution of cocaine which was applied with cotton to the pharynx. However, the repeated mopping of the throat was much more disagreeable for the patient because there was more retching and, since it gave no better anesthesia than is obtained by the present method, the older one was given up. Between the sprayings with pontocaine we talk to the patient about things apart from the examination and we try to maintain a manner which will lessen any apprehension which the patient may have. For the same reason an operating room is not used and as a rule we do not wear a gown, mask or gloves; these all connote to many a major procedure and they are usually unnecessary. The "medical asepsis" is simple cleansing of the hands with a bristle brush and soap and washing the gastroscope with soap and water and alcohol.

About five minutes after the second spraying of pontocaine the patient is asked to sit on the edge of an examining table and an Ewald-Boas tube (No. 30 French) is passed. With the tube

in place the patient lies on his left side, an orderly raises the foot of the table and the tube is slowly withdrawn. This procedure assures us of the patency of the esophagus and we remove most of the gastric content. The table is then lowered. The patient continues to lie on his left side with his left arm bent at the elbow and under his body and the knees are drawn up. His head is held between the hands of an orderly or it is rested on a hard pillow of adjustable thickness which our nurse has devised. With the head in a natural position the 'scope is passed gently, slowly, and by touch alone almost to the mark which indicates the cardia. Then the head is extended on the neck and the 'scope is introduced into the
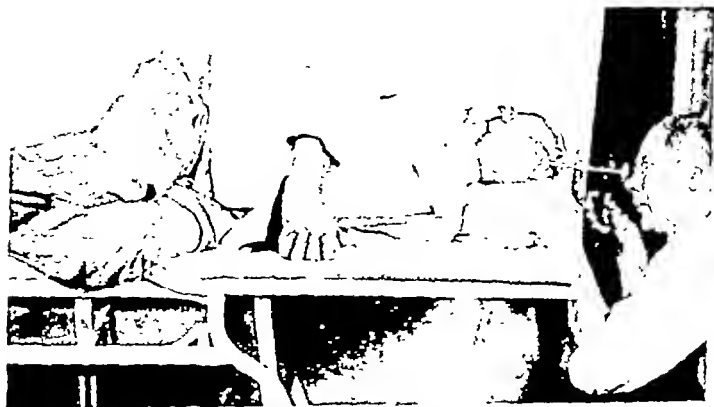
Fig. 53.—The position for gastroscopy.

stomach (Fig. 53). Some resistance is commonly encountered at the level of the constrictor of the pharynx and at the cardia but with patient waiting this soon disappears. The patient may be reassured that the little discomfort that passing the cardia causes is quite usual and not alarming. A little air is blown into the stomach and the 'scope is advanced until the musculus sphincter antri is seen. This is the sickle-shaped curtain at the entrance to the antrum. We use minimal inflation at first and we have learned that patience and waiting often bring the pylorus into view when ballooning the stomach with more air makes it more difficult to see. Only after the pylorus has been inspected is more air forced in and the walls

of the stomach inspected as the 'scope is withdrawn. After the procedure the patient lies down for an hour and then he is allowed to carry on as before the examination. For any traumatic sore throat he is advised to use a saline gargle.

Findings and Their Interpretation.—Now what can we see with the gastroscope? In the first place, we can often see benign and malignant *ulcerations, tumors* of the stomach, and *foreign bodies,* and there is no other way in which *gastritis* of any type can be so satisfactorily diagnosed. Indeed, it does seem to us that a little too much stress is laid on gastritis of a mild grade as a possible cause of symptoms. Those symptoms may be on a functional basis and the gastritis may be only an incidental finding, as sinus disease may be only an incidental disease or condition in a person who has chronic headaches. The very finding of a little redness of the mucosa doesn't mean that the redness represents the cause of a dyspepsia.

At this time we are most enthusiastic about the positive gastroscopic findings when they represent gross lesions, particularly ulcerating lesions. Bitter experience has made us hesitant about declaring a stomach to be normal in the face of direct roentgenographic evidence of disease. Sometimes we are correct and the skiagrams are wrong, but again the gastroscopist may miss some lesions that roentgenograms show. The gastroscope is a most valuable adjunct to the x-ray in the diagnosis of neoplasms and ulcers of the stomach but it by no means replaces the x-ray. If we had to dispense with either gastric roentgenography or with the gastroscope we would give up the latter, but it would be with reluctance.

Other Tests.—Of course there are many other tests for gastric disease. One may determine the nonprotein nitrogen content of the gastric juice; he may determine the excretion of a dye into the gastric lumen; he may study the carbon dioxide combining power of the blood plasma in cases of pyloric obstruction to learn of the loss of chlorides; he may even wish to look for blood on a swallowed string. These are accessory examinations and one must not be misled by the lesser methods of examination. They are most useful when they confirm the results of the methods which we have discussed.

THE CLINICAL APPLICATION OF OUR TECHNICS

Our goal in the discussion of the technic of the gastric examination is the application of these technics in the study of patients and the formation of the correct diagnoses. Let us review critically two cases in which these technics were used.

Case I.—Mr. R. W. (J. H. H. No. 185451) a locomotive engineer, fifty-four years of age, consulted us in September of 1939 because he had had pain under his left costal margin for two or three months. Prior to May or June of 1939 he had been in excellent health except for diarrhea of unexplained origin for six years after living for a year in Brazil. That was nineteen years before the onset of the symptoms for which he came to Baltimore. In the summer of 1939 he had been worried and a little depressed because one of his close friends, another locomotive engineer, had died following an operation for carcinoma of the colon in Baltimore. It was during this period of nervous strain and two or three months before he presented himself to us that he first had pain under his left costal margin. It would waken him at night and it would annoy him just before his noon meal. The pain would be relieved by soda but not by food. In fact, he was afraid to eat. Though his appetite was excellent, he felt better when taking soft food.

Because his complaints persisted his family physician sent him to a hospital for study. There his examinations (including roentgen studies of the stomach) were negative except for hyperacidity. He was given an ulcer diet and aluminum hydroxide which relieved his symptoms. Naturally when they had no real evidence of organic disease, the consultants reported to his doctor, "He is very much worried about carcinoma since the death of Engineer ————, his close friend." However, the patient had the feeling that all was not well in his abdomen and he came to Baltimore.

His physical examination revealed nothing except moderate arteriosclerosis. There was a scar of an appendicostomy.

The gastric acids after an Ewald test-meal were 48 and 66. The hemoglobin was 90 per cent. Two stool specimens showed only traces of occult blood to the benzidine test. The serologic test for syphilis was negative. Fluoroscopic examination of the stomach and duodenum was negative except for some irregularity such as might be caused by large rugal folds high on the greater curvature. To our surprise, gastroscopic examination revealed an ulcer on the anterior wall near the greater curvature; the rugae around it were very coarse and there was a definite gastritis around the ulcer. The patient was sent home on ulcer therapy.

At our direction, he returned October 11, 1939, saying that he had had some distress under the left costal margin that had been relieved by drinking goat's milk so that there had been absolutely no distress for a week before his second visit here. He said that he felt like returning to work. He weighed 164¼ pounds and his abdominal examination revealed nothing.

The hemoglobin reading was 94 per cent. A specimen of stool on a meat-free diet gave a 4+ reaction when tested for occult blood with benzidine. Gastroscopic examination revealed the same ulcer in the same location and we remarked that it looked more "angry" than it formerly appeared. The patient was sent home to continue his ulcer regimen and to return in three weeks.

On November 6, 1939, we saw the patient again. He had gained two or three pounds in weight. He had had increasing distress under the left costal

margin which food and alkalis did not relieve and for which he had taken
injections of Larostidine at the hands of his local physician without benefit.
His appetite was excellent. His physical examination was negative.

The hemoglobin was again 94 per cent. The stool gave a 3+ to 4+ re-
action for occult blood. Gastroscopic examination revealed the ulcer crater
with the marked gastritis around it and we remarked that the lesion looked
like a malignancy. The patient was admitted to the hospital for surgical ex-
ploration and there he was re-x-rayed. The rugal folds were very heavy and
the first roentgenologist diagnosed large rugal folds which probably represented
only a gastritis. Alarmed by this reading of the films (Fig. 54), and fearing
that we would be the laughing stock of the hospital after the exploration, we
passed the gastroscope a fourth time and we again saw the lesion. Later that
same day a second roentgenologist reviewed the films, asked for a clinical
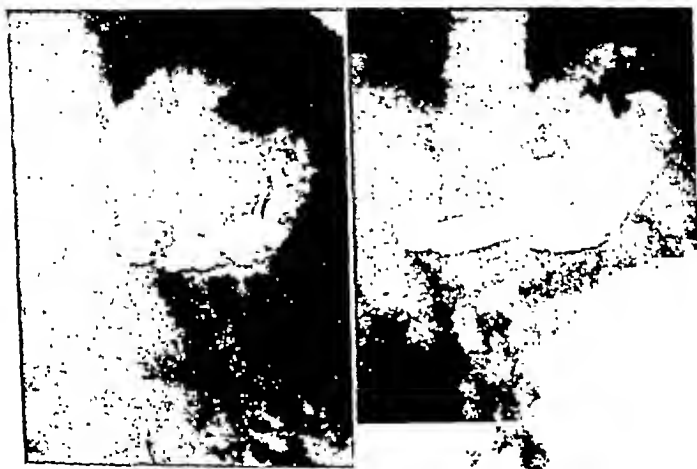
Fig. 54.—Showing defect at cardiac end of stomach.

history before making his report, and said, "Defect on the posterior wall of
the cardiac end of the stomach which is due to an ulcerative lesion."

On November 15, 1939, Dr. William F. Rienhoff explored the abdomen and
resected the distal four fifths of the stomach. On the greater curvature was
an ulcer crater the size of a half-dollar and sections of the ulcer showed it to
be malignant. There were no evidences of metastases and the patient was
digestively well when seen in September, 1940.

This case illustrates the value of the history of persistent
symptoms, the value of stool studies for occult blood, and the
value of the gastroscopic findings in the face of a negative
physical examination, equivocal roentgen studies, and plenty
of hydrochloric acid in the stomach. Without the gastroscopic

evidence of ulceration we would probably have temporized until the growth had become so extensive that a cure would not have been possible. The following case illustrates that very point:

Case II.—Mrs. G. S. (J. H. H. No. 207745) was first seen in August of 1938 when she was sixty-four years of age and when she complained of ringing in the ears, deafness, "indigestion" of years' duration, nervousness, and dull headache. This galaxy of complaints, particularly the long-standing dyspepsia, suggested functional trouble. This concept was re-enforced when we learned that her husband had died the preceding February after a long illness that had been a great strain on the patient, and that with the menopause thirteen years before her consultation with us she had had a "nervous breakdown" and "nervous indigestion." She had lost no weight.

Fig. 55.—Normal appearing stomach and duodenum in Case II, May, 1939.

The patient weighed 95 pounds; she was markedly arteriosclerotic; and she had a ptotic, thin-walled abdomen in which the right kidney could be easily palpated. Laboratory tests, that did not include an examination of the feces, were all negative. An otolaryngologist thought that the tinnitus was the result of degenerative changes in the ears (though she hasn't complained of it for at least a year and a half).

In December of 1938 the patient weighed 97 pounds and she had been subjectively helped by belladonna, phenobarbital, and bowel management.

In April of 1939 the patient complained of epigastric burning, some lower abdominal pain, and a little constipation. She had gotten to 100 pounds but at the examination she weighed 98 pounds. The blood and urine showed nothing. Roentgenograms were not made. She was given a bitter tonic and an attempt was made to manage the bowel without irritant laxatives.

On April 28, 1939, the patient had a hemorrhage from the upper gastro-

intestinal tract and, though she did not vomit blood, the stools were tarry. Two days later she had another similar hemorrhage. With recovery from the bleeding the patient had a Graham gallbladder series that was negative; a normal colon was found by barium enema; and the stomach and duodenum were said to be roentgenologically normal (Fig. 55). Gastroscopic examination revealed a mild atrophic gastritis. With my tongue in my cheek I answered the queries of the patient and her family with, "The bleeding probably came from a gastritis."

In August of 1939 the patient weighed 100 pounds and she reported that she was really very well. Her hemoglobin was then 98 per cent.

In October of 1939 she weighed a little over 100 pounds and her bowels were moving well with nightly retention enemata of cotton seed oil. She had no complaints. Unfortunately roentgen studies were not repeated.

The patient was not heard from again until August of 1940. In June she had begun to have some vague dyspepsia with abdominal distention, constipation, and protruding hemorrhoids. When seen in August she looked pale;
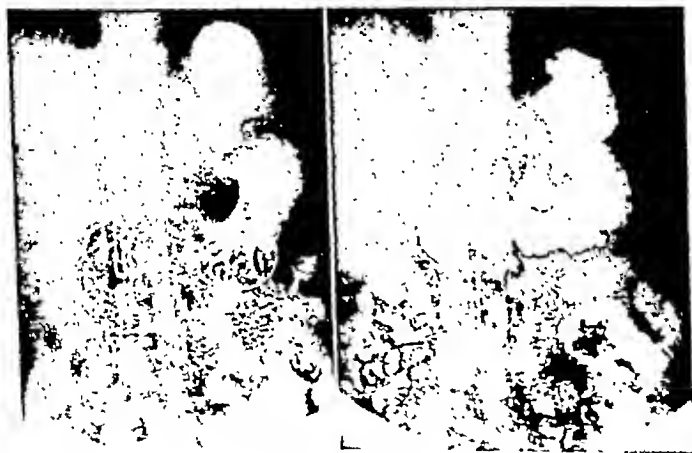
Fig. 56.—Films in Case II, August, 1940.

the abdomen was distended; the navel was somewhat flattened and there was a prominent hypogastric vein in the right lower quadrant; there was possibly some ascites. The patient was sent to the hospital. There her weight was 95 pounds. The hemoglobin was 64 per cent. Tests of the stool for occult blood were strongly positive. After an alcohol test-meal the gastric acids were 0 and 8; forty minutes after histamine stimulation they were 9 and 35. The serum bilirubin was 1.0 mg. per cent.

Fluoroscopic examination by the first observer showed a defect high on the greater curvature of the stomach and he felt that this represented a carcinoma. Films were made and some of them are shown here (Fig. 56). After he had seen these skiagrams the observer thought that the defect might have been caused by external pressure. He had a barium enema given and the defect was apart from the colon. A second roentgenologist reviewed the films and declined to come to a conclusion until he had made his own roentgeno-

scopic examination and more films. His report finally was: "Filling defect on the lesser curvature and posterior wall of the stomach due to the presence of a neoplasm." Gastroscopic examination was done with the greatest ease. There was an area of inflammation on the anterior wall at the cardiac end of the stomach and some irregular infiltrations on the lesser curvature with some erosions but the purple ulcerations of the usual carcinoma were not seen. Too late we knew that Mrs. G. S. had a gastric carcinoma.

Looking backward, what was wrong with our management of this second case? Did we fail to diagnose the cancer because we had poor technic in our tests? I doubt that our technic was too bad. Perhaps more careful study by relief methods would have demonstrated the lesion in the spring of 1939. However, careful scrutiny now shows that the films made in April of that year reveal the slightest mottling on the greater curvature. In anyone else this would be called a rugal fold and it was so termed in 1939. However, we now know that in this patient it represented organic disease. What was the matter with our gastroscopic technic in the spring of 1939? Why didn't we see the lesion then? Probably we missed it because it was then confined chiefly to the posterior wall of the stomach rather high in the cardiac portion and this region is not well seen with the usual gastroscope. Indeed, a year and a half after the initial symptom of hemorrhage the gastroscopic evidence of malignancy wasn't too striking.

What things were "left undone . . . which we ought to have done"? Certainly we should have had the stools tested for *occult blood* in the summer and autumn of 1939. They surely would have been positive and, following that lead, roentgenograms should have been made again. It is difficult to insist that persons have a roentgen examination repeated when it has recently been done with supposedly normal findings, especially when they are in modest financial circumstances and when they feel well. However, when occult blood is persistently found in the stools and when there is no satisfactory explanation for its presence, such an examination is justified, particularly in elderly persons.

In this latter case "we have done" one thing "which we ought not to have done"; we were satisfied with the diagnosis of "hemorrhage from gastritis." We forgot the dictum: "Look for ulcer or cancer in all cases of gastritis!"

After all we are doctors, not technicians. Excellent though

the technic of the gastric examination may be, even better must be our judgment in applying and interpreting the few important tests for gastric disease, or an important and perhaps serious gastric condition may be missed.

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GENERAL ANESTHESIA IN HOME AND OFFICE PRACTICE

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For many years after the discovery of general anesthesia in the middle of the last century, ether, chloroform and nitrous oxide were the only agents used. During the past two decades, many new anesthetic agents have been added to our armamentarium. The general practitioner very wisely allows these new drugs to be tried and proved in the larger hospitals under supervision of experienced anesthetists before trying them himself. Several of these new drugs will be presented as particularly adapted to office and home practice.

A WIDER USE OF GENERAL ANESTHESIA IS INDICATED

There is no branch of medicine in which there is required more flexibility of action and this under more trying conditions than that of general practice. The general practitioner's lot is a hard one even when he has easy access to a modern hospital, to say nothing of the hundreds who do not have hospitalization within reach of their patients. For each practitioner, in and close to a city, who can readily take his patients to a hospital, there are many in the country and smaller towns who cannot. When time counts, the hospital may be too far away and the emergency may have to be dealt with on the spot. Such circumstances warrant the employment of general anesthesia by the general practitioner, beyond the shadow of a doubt. On the other hand, many conditions present themselves when a hospital is within reach, but for very good reasons it may be more advisable to handle the situation in the office or the home under general anesthesia by the physician in charge.

No doubt in its early use, general anesthesia was adminis-

tered more in the office and the home than it was in hospitals. Fifty years ago there were probably more physicians in general practice than in hospital practice who were adept in the use of chloroform anesthesia. At the present time, there are many of the older general practitioners who could teach the specialists in anesthesia a lot about the use of chloroform; in fact, few specialists today know anything at all about administering chloroform anesthesia.

In many small hospitals, the situation is commonly accepted wherein the general practitioner administers the anesthetic to his own patients for major operations of lengthy duration; these opportunities are not frequent enough to make him an accomplished anesthetist, and no general practice is large enough to offer the physician sufficient experience when administering the anesthetic only to his own patients. When anesthesia is given in the home or office it is of short duration and usually relaxation is not necessary in order to perform the operation satisfactorily. Short anesthesias for minor operations in the home or office are safer than longer anesthesias for major operations in the hospital when given by the same person of little experience. The fact that the operation is being performed in a hospital does not insure greater safety to the patient unless there is available in that hospital experienced supervision.

Certainly there is no discovery in medicine affording a greater boon to humanity than general anesthesia. Before anesthesia, many patients died from the shock of the operation alone. Many patients today undergo minor operations without anesthesia simply because they are able to take the pain. Those who are expected to or do make a fuss enjoy the benefits of anesthesia. The element of *shock and pain* is often minimized by the operator, and the patient, between his outcries, is even told that the doctor is not hurting him. Such procedures, with few exceptions, are unnecessary and near barbaric. No patient facing a painful operation should be denied the benefit of anesthesia if it can possibly be given. "An operation exceedingly painful, if continued without intermission for some time, will cause death by exhaustion of the nervous powers. Of this we have numerous instances." (Bouisson, 1850.)

Every general practitioner may not desire to use general anesthesia in the office, but all who do obstetrics in the home must use it in one form or another. Some may feel that their lack of experience forbids the administration of even short anesthesias; this must be settled by the individual. It seems to me the personal equation enters in this manner: If a man tries, but cannot safely drive an automobile, then he should let someone else drive for him; if a physician attempts to handle all the fractures coming into his hands, but consistently gets poor results, he should turn them over to another more adept than he in this field and content himself to give only the preliminary treatment of fractures. The same applies to the use of general anesthesia by the practitioner. If he consistently encounters trouble, he should employ the services of someone more successful; this will result in better care for his patients and also greater peace of mind for himself.

EARLY AND MODERN METHODS

Before general anesthesia, many drugs were employed in an effort to render the patient insensible to pain. Hyoscyamus, conium, Indian hemp and other sedatives proved to be of little value. Exposure to cold to the extent of half freezing the patient was tried for amputations, with poor results. Gangrene usually supervened and pneumonia was a frequent complication. Drunkenness induced by large quantities of alcohol was practiced with little success. After the operation was finally accomplished, there would be injured assistants as a result of the drunken man's lack of cooperation.

The early method of general anesthesia was one of *force and speed*. A cone, properly made for suffocating the patient, was almost filled with cotton or gauze, and into this was poured a liberal portion of chloroform or ether; then with attendants restraining the patient and the cone clamped tightly over his face, he was quickly rendered unconscious. Anesthesia resulted from a combination of the anesthetic agent plus asphyxiation. Patients with bad hearts were placed in jeopardy, while small children were in danger of dying from fright.

The perfected technic of administration today, together with improved agents, has done much to remove the dread formerly associated with anesthesia. Preanesthetic narcosis

leaves no memory of the actual induction, or at most, a very vague one. Instead of the choking, concentrated induction with ether, the drop method is used. In a general practice where there is sufficient home or office surgery to warrant it, nitrous oxide-oxygen induction can be employed to advantage. This, of course, necessitates considerable familiarity with the use and administration of nitrous oxide, and possession of a modern, portable gas machine. While nitrous oxide-oxygen anesthesia is more difficult to give from the standpoint of safety to the patient, there is no reason why the general practitioner should not employ it, for short operations, with the same success as do the dentists.

It would seem that in a practice where opportunities for using general anesthesia present themselves frequently enough, it would be just as feasible for a visiting physician to take a short course in anesthesia as it would be for him to brush up on any other phase of medicine. Then, too, in some localities, it might be ideal for one physician to rather specialize in anesthesia in conjunction with his general practice. With the present-day rapid transportation, he could cover quite an area, assisting other physicians in this capacity. Certainly, with the present trend toward the building of small hospitals in rural communities, a man thus equipped and established would be a valuable asset to that locality. A young man fresh from his hospital experience would be the ideal one to take a portable gas machine to his new location and let it be known to his contemporaries that he is available for this service. Such cooperation would hardly result in the encroachment of the anesthetist on the operator's practice.

The two most important *fundamentals* in the modern administration of anesthesia are:

1. *Slow, smooth induction.* The anesthetic should not be pushed to the point of causing choking or struggling. When dealing with adults, a slow induction is more satisfactory to all concerned. When dealing with children who are crying and have to be restrained, a rapid induction is justifiable and more satisfactory, but care must be taken lest they suddenly become overanesthetized from the deep inhalations between outcries.

2. *Point of safety.* When judging the *depth* of anesthesia,

one should always err on the safe side. If you are in doubt as to the depth of anesthesia, stop and wait until there is definite evidence that the patient is not deeply anesthetized. When using nitrous oxide-oxygen anesthesia, one is limited in the depth to which it can be carried. So long as the patient is getting enough oxygen to maintain a good color, there is no danger; the danger period arrives when the mixture becomes too lean of oxygen and cyanosis occurs.

One should guard against the feeling of having attained proficiency after giving a few dozen anesthesias. Usually the intern, after giving a few anesthesias without mishap, feels it is a very simple procedure, when in fact, if thrown on his own resources, he would not be able to cope with many of the difficult situations that may arise during the course of anesthesia. To be careless in administering an anesthetic is to betray a trust, for if the patient did not trust the anesthetist, he would never consent to accepting it. The *safe* anesthetist is one who, far from knowing it all, may often be in doubt, yet always plays safe when determining the depth of anesthesia. Of course, smoothness and depth of anesthesia do not play as important a part in office practice for short operations as in the hospital for long and more serious ones. Office equipment for anesthesia need not be as elaborate as that used in the hospital. For only short anesthesias, a gas machine, if one is used, need not be of the rebreathing type using a soda lime filter. In short, very little ingenuity is necessary to devise equipment for administering anesthesia in the home or office.

VARIOUS AGENTS AND ADMINISTRATION

Not so many years ago, there were only three agents available for office anesthesia—chloroform, ether and nitrous oxide-oxygen. Now they are numerous; each year some new drug is put on the market, and some of these are found by trial to be of value in the hands of specialists, others are tried and soon discarded, while many do not have sufficient merits to receive a trial. Of the various agents, the following are the most commonly used today:

1. Nitrous oxide-oxygen.
2. Ether.
3. Chloroform.
4. Vinethene (Divinyl ether).
5. Cyclopropane.
6. Ethylene.
7. Sodium pentothal.
8. Evipal.

For preanesthetic narcosis:

1. Avertin.
2. Nembutal, seconal, sodium amytal, etc.

Of these agents, the most adaptable to home and office use could be listed in the following order with regard to safety and ease of administration:

1. Ether.
2. Chloroform.
3. Vinethene.

4. Sodium pentothal, Evipal.
5. Nitrous oxide-oxygen.

Premedication.—While premedication is not always possible or necessary, it makes for smoother induction. *Atropine*, grain $\frac{1}{120}$ hypodermically, should be given to all adults. This quite materially reduces secretions and amount of mucus in the throat. *Morphine* may also be given unless a preanesthetic narcotic is used. Avertin for office practice would not be advisible. *Nembutal* or *seconal*, grains 3 by mouth to adults forty-five minutes before operation, allows smoother induction and administration. Very often this dose is sufficient to induce deep sleep, on top of which the anesthetic may be added without resistance; very seldom does the patient have any memory of induction.

ETHER

Of all the anesthetic agents preferred today, there is none safer or more satisfactory for all-around use than ether. It would be the choice of every anesthetist in the country if he had to use one agent for all conditions. As it is the safest agent in the hands of the most experienced, it therefore must be the safest in the hands of the inexperienced. If it were not for the disagreeable odor and postoperative nausea and vomiting associated with it, one would need look no further for a better anesthetic.

Except in the presence of pulmonary or upper respiratory infections, ether can safely be administered in almost any condition, and especially when the anesthetic is light. As with any other anesthetic agent given to a patient not properly prepared, there is always the danger of *aspiration of vomitus.* This is probably the greatest risk encountered in short, light,

emergency anesthesias. All other risks are practically nil if the ether is discontinued as soon as relaxation is effected.

Administration.—An open cone or improvised frame mask of wire, over which 6 to 8 layers of gauze or a washcloth are placed, is all the equipment necessary for administration. About half a teaspoon of vaselin should be placed over each closed eye and over this a piece of protective rubber. These rubber strips can be cut from the cuffs of old rubber gloves.

The *open drop method* is to be preferred to that with the closed cone. With the mask gently in place over the patient's face, drop the ether at a rate not exceeding 15 drops per minute for the first two or three minutes. Slowly increase the rate until the mask is kept wet. When respirations become free and easy and the patient is relaxed the anesthetic may be discontinued; there should be sufficient time to perform the operation or manipulation usually done in the office, without additional ether.

Complications and Their Management.—If at any time during the administration the patient should *retch*, the mask ought to be raised each time the patient gags. If *vomiting* occurs, the mask must be removed, the head and shoulders lowered, if possible, and the face turned to one side. In this way, the patient will be allowed to get rid of the vomitus with very little danger of aspiration. Very few patients under anesthesia light enough to allow vomiting will aspirate if the face is uncovered and the vomitus is allowed to escape. The administration of the anesthetic should not be resumed until vomiting has ceased, even though the patient regains consciousness while waiting.

If, during induction, *the patient's jaws become clenched* and the exchange is so poor that cyanosis ensues, do not try to pry open the jaws with any instrument, for you may push out teeth or damage the lips or gums. All that is needed is a small rubber tube about 8 inches long and a little larger in diameter than a lead pencil, with one end cut on the oblique and several small holes punched in the sides. Insert the pointed end in one nares to a depth where breathing takes place through the tube. If such a tube is not at hand, extend the head and insert the index finger of each hand along the outer margin of the teeth as far back as possible, care being

taken not to get the fingers between the upper and lower teeth, should the jaws separate. This position of the fingers creates an air space through which the patient may get a sufficient exchange. The curved, metal airway is of no use when the jaws are clenched, since it cannot be inserted. When anesthesia becomes sufficiently deep, the jaws will relax and breathing will become free and easy.

If the operative procedure is so long that continued administration becomes necessary, the *pupils* and *respirations* must be closely observed lest the patient become too deeply anesthetized. For the patient to begin to awaken and squirm is inconvenient but safe; for him to reach deep anesthesia is dangerous. At any time the pupils react to light, the patient is not deeply anesthetized. Whenever pupils, having reacted to light before, cease to do so, the patient is under deeper anesthesia than necessary and cessation of anesthesia is indicated. The size of the pupils is not accurate as a gauge, since during the excitement stage they may be as large as during profound anesthesia.

It is far better to learn to judge the depth of anesthesia by the character of the *respirations* than by pupillary reflex; especially is this true when the operator must use a layman to continue the anesthesia while he operates. The operator does not have easy access to the pupils, but he can note the type of respiration while operating. When inspiration is free and easy, the patient is safe. If inspiration becomes short and jerky, the anesthesia is most likely deep and should be stopped and not resumed until inspiration becomes longer. Difficult inspiration due to a sagging tongue is not to be confused with that from deep anesthesia. The nasal tube will take care of the obstruction caused by the tongue.

Some offices are equipped with suction or a suction machine. While not entirely necessary, suction frequently is advantageous in relieving the patient of mucus that may collect in the mouth and throat. Vomitus is usually too heavy to be drawn off by suction.

During induction, patients will frequently hold their breath to a point of deep *cyanosis*. While this condition is rarely serious, it might readily become so in the presence of a weak myocardium. Usually the passing of a nasal tube will cause

gagging and resumption of breathing. It is very convenient to have a small tank of oxygen handy when cyanosis occurs. Should cyanosis occur in a child from laryngospasm, making inspiration labored and insufficient, the anesthetic should be discontinued until normal breathing is resumed.

CHLOROFORM

While chloroform is still preferred by many general practitioners, it is not as safe as ether in the hands of one inexperienced in the use of both. It is quicker and pleasanter to the patient, but the margin of safety is narrower. Much less chloroform than ether is required for the same duration of time and depth of anesthesia. Though chloroform is less irritating to the respiratory tract, it has definite toxic effects on the heart and liver and therein lies the danger. A combination of chloroform for early induction followed by ether for relaxation is not a bad one. With this method, one is less likely to incur resistance, nausea and vomiting. When giving chloroform to crying children, care must be taken lest they receive an overdose.

NITROUS OXIDE-OXYGEN

Nitrous oxide anesthesia should not be given by an inexperienced person. Supervised experience in a hospital is a prerequisite to the use of this type of anesthesia. There is just as much danger the first few minutes as there is during the latter part of administration. There is one important thing to remember about nitrous oxide anesthesia, namely, that so long as the patient is getting enough oxygen to maintain a good color, there is no danger whatever. Beginning *cyanosis* is the danger signal. If proper relaxation cannot be effected while keeping the patient a good color, changing the mixture to produce cyanosis not only increases rigidity from asphyxiation but increases the hazard. A tinge of cyanosis is permissible, but definite blueness is not.

Because of the cumbersome equipment necessary, nitrous oxide-oxygen anesthesia is not practicable for use by the general practitioner in the home. One cannot administer nitrous oxide safely and operate at the same time. Most dentists have discontinued this dual performance and now employ an as-

sistant to conduct the anesthesia. There are many conditions in general practice favorable to the use of nitrous oxide if there is available someone, preferably a physician, who is experienced in its use. Most notable of these conditions is during labor when quick analgesia is desirable with pains, without effecting complete anesthesia.

VINETHENE (VINYL ETHER)

Vinethene consists of about 96 per cent pure vinyl ether with 3.5 per cent absolute alcohol and an oxidation inhibitor.

Induction with vinethene is quicker than with ether and about as quick as with chloroform. Vinethene is packaged in small bottles of various sizes, allowing the use of a bottle estimated to be the amount necessary for the operation to be performed. This prevents waste, since one should never use a bottle that has been opened any length of time.

Vinethene should be used only for *short* anesthesias. When used for long periods of time, it has caused focal necrosis of the liver similar to that sometimes seen after chloroform anesthesia. The awakening period following vinethene is very short, and this is of particular advantage when the patient must be attended in the office until fully conscious. Nausea and vomiting following vinethene are rare in a patient properly prepared beforehand. With crying children, the same care must be taken as when using chloroform.

SODIUM PENTOTHAL AND EVIPAL

The composition of these two drugs, together with directions for mixing and administration, accompany each package. Both are given intravenously and both have about the same potency, action and safety.

Sodium pentothal comes with two 20 cc. ampules to the package. One ampule contains 20 cc. of distilled water and the other 1 gm. of powdered pentothal. This dilution is preferable to the original 1:10, since the weaker solution has less tendency to cause thrombosis at the site of injection.

Pentothal should not be given to small children; preferably ten years and not under six years should be the lower limit. Instead of 1:20, a 1:40 solution is better in children. The more dilute solution gives greater safety and just as good results.

Administration.—One sterile 20-cc. syringe with needle
is all the equipment necessary for administration.   It is ad-
visable, however, to use a small hypodermic syringe to put a
drop of 0.5 per cent novocain in the skin at the site of injec-
tion. This renders the injection of pentothal absolutely pain-
less while using a needle of 18 gauge on the 20-cc. syringe.
After 3 cc. of pentothal have been injected, a minute should
elapse before running in the anesthetic dose. With this pre-
caution, any possible idiosyncrasy to the drug may be de-
tected, though none of any consequence has been noted as yet.
The patient should then be instructed to count slowly while
the solution is run in at the rate of 1 cc. every five seconds.
When counting ceases, inject about two-thirds the amount

Rubber nasal tube

Metal air-way

Fig. 57.—Rubber nasal tube and metal air-way.

already used, withdraw the needle, and proceed with the op-
eration.

Unless a rubber catheter is passed through the nose or a
metal airway is put in the mouth (Fig. 57), there should be
someone present to hold the patient's chin up to insure a free
exchange. The anesthesia thus obtained will suffice for many
operations of ten to fifteen minutes' duration. The operation
may continue for some time after the patient becomes restless,
without any remembered pain. If it should become necessary
to prolong the operation beyond the duration of the anesthesia,
an additional dose may be given from the same syringe which
has been kept sterile.

The most disconcerting phase of the use of pentothal is
the speed of its action in producing sleep. When using it for

the first time, one is inclined to stop the injection when counting ceases and hope only that the patient will awaken. After using it or seeing it used several times, one can give it with confidence and composure. The usual dose will run from 8 cc. to 10 cc. in adults, though in large, husky males 12 cc. or 13 cc. may be necessary before sleep is effected.

Complications.—*Nausea* and *vomiting* seldom follow the use of pentothal when given to a patient with an empty stomach—and it should never be given unless the stomach is empty. The danger of aspiration is much greater than when some type of inhalation anesthesia is used. This danger can be avoided, however, by passing the stomach tube before giving pentothal.

After pentothal anesthesia, the patient will require attendance for twenty or thirty minutes and will be able to be assisted from the office within an hour. The recovery time is less than after ether, chloroform or vinethene. The anesthesia is so pleasant to the patient that one would have difficulty in giving him anything else should a subsequent operation be necessary.

THE CHOICE OF ANESTHETIC

Some operations presenting in general practice and the anesthetic of choice follow:

Fractures:

1. Sodium pentothal when the patient's stomach is properly emptied.
2. Chloroform, ether, or vinethene. Chloroform only when the physician is familiar with its use.

Incision and Drainage of Abscesses:

1. Nitrous oxide-oxygen if practicable.
2. Chloroform or vinethene.
3. Sodium pentothal.
4. Ether.

Suture of Cuts and Lacerations:

1. Sodium pentothal.
2. Vinethene.
3. Chloroform.
4. Ether.

Paracentesis:

1. Vinethene.
2. Chloroform.
3. Ether.

Obstetrics.—The general practitioner probably uses anesthesia in obstetrics more than in all other conditions combined. Nitrous oxide-oxygen should be used only when there is another physician present who knows how to administer it. Of all the anesthetic agents, there is none more satisfactory for home obstetrics than chloroform.   The favorable action of chloroform over ether counteracts any relative difference of safety. Due to the various conditions and lack of assistance, the physician must supervise the anesthesia from start to finish and at the same time conduct delivery. Powerful narcotics should not be used as anesthetic agents because of the resulting narcosis of the baby. Ether does very well for the actual delivery, but not for dulling the intense pains preceding the delivery. After all is said and done, most specialists could take lessons from many general practitioners in the conduction of anesthesia during delivery in the home.

PRECAUTIONS AND STIMULANTS

1. The patient's stomach should be empty either from abstinence from food and drink or use of the stomach tube.

2. Atropine, 1:150 to 1:120, should be given to adults a half hour before operation.

3. Preanesthetic narcosis, while optional, is desirable in many cases.

4. A nasal tube or airway to insure free exchange should be available.

5. Sufficient assistance should be at hand before the anesthesia is started.

6. Should vomiting occur, quickly turn the patient's head to one side and lower the head and shoulders if possible.

7. Coramine or metrazol should be kept handy. Not less than 3 cc. of either should be given intravenously when stimulation is indicated.

SUMMARY

Anesthesia in general practice is not only indicated but has far more use than it is being put to at the present time.

With the use of intravenous anesthesia, the operator can administer the anesthetic and have a longer time to perform the operation.

There is a field in many communities, with small hospital facilities, for a physician to do expert anesthesia work along with his general practice.

Mutual assistance among neighboring physicians in the rural sections would add greatly to the safety and ease of minor operations performed by the general practitioner under general anesthesia.

Every general practitioner should be able to administer safely some anesthetic agent.

Excepting those who are well able, patients should not have to incur hospital expenses for conditions that can easily be taken care of in the office or home.

Operations requiring prolonged, continuous anesthesia should not be undertaken under the dual role of anesthetist and operator; proper assistance should be arranged for beforehand.

PRACTICAL APPLICATION OF LIVER FUNCTION TESTS

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The Place of Liver Function Tests.—Liver function tests have a definite place in the diagnosis of hepatic disturbances, in the confirmation of liver dysfunction, and as guides to both prognosis and to therapy.

Such recognition has been delayed by two factors:

1. The experiments of Mann revealed normal function upon the excision of seven tenths of the dog's liver, hence it was inferred that almost the entire liver must be destroyed before such changes are to be detected by liver function tests and that the latter must therefore be of little or no practical aid. Such reasoning seems fallacious because in these experiments the major portion of a *normal* liver was excised and the residual tissue was normal. This is not analogous to the clinical situation: (*a*) When hepatic disturbance occurs, all of the cells engaged in a particular activity or interdependent activities are concurrently affected. (*b*) It is a fact that liver function tests not infrequently manifest hepatic dysfunction without any other objective evidence of such.

2. Too often, one test measuring a specific function of the liver was expected to measure others. Thus, when the liver excretory mechanism is relatively intact and there is a disturbance in its detoxifying power, bromsulfalein which gauges only the former cannot be expected to detect the latter which is satisfactorily done by the hippuric acid test. Too often one test denoting the impairment of a specific function under one circumstance was expected to show the same aberration under another. For instance, the galactose tolerance test is frequently positive in early, acute intrahepatic jaundice because

the abrupt disturbance in the carbohydrate function of the liver interferes with adequate absorption of this product. Later, as well as in not well advanced chronic liver disease, this organ has had opportunity to compensate sufficiently to render the test negative. In still later stages it may become positive again when the glycogenic function fails. Here liver disease is constantly present but varying conditions alter what is to be expected from the test.

Failure to recognize the aforementioned considerations resulted in inevitable disappointments which have been erroneously ascribed to the tests.

The testing of multiple functions requires the use of multiple tests. In our hands one or more of these has been positive in the great majority of those with suspected or actual liver disease. Had we used but one test measuring one function the results in many would have been negative. Appraisement of the progression or regression of the liver disturbance as well as the response to therapy requires the serial or repetitious determinations of these functional tests.

In general, the value of liver function tests will depend on a knowledge of the indications and limitations of the several available practical procedures, the nature of the function to be tested, and the intelligent interpretation of such information in association with all other data in the case.

Types of Liver Function Tests.—A simple classification by MacLagen, which I have modified, of the more commonly used tests follows:

A. Tests Depending upon Excretion of Bile:
    Van den Bergh Reaction—Icteric Index.
    Azorubin S Test.
    Bromsulfalein Excretion Test.
    Bilirubin Excretion Test.
    Serum Phosphatase and Cholesterol Estimations.
    Examination of Duodenal Fluid.
    Cholecystography.
B. Tests Not Depending on Excretion of Bile:
    Deaminating Function (*estimation of blood amino acids*).
    Detoxicating Function (Hippuric acid and *Salicyluric acid*).
    Glycogenic Function (*glucose*, levulose, galactose tolerance).

Those in italics are not of generally recognized practical value.

In our experience the concurrent use of the quantitative Van den Bergh reaction, the bromsulfalein and the hippuric acid tests has been found to be the most practical and satisfactory means of determining liver function, except in the differential diagnosis of acute jaundice. In the latter, the concurrent repetitious use of the galactose tolerance test, the cholesterol partition, the blood bilirubin, the phosphatase level of the blood and the determination of urinary urobilinogen have been the most helpful.

The physiology and technic involved in the various liver function tests referred to will not be presented here. They are adequately discussed in recent works on clinical laboratory procedures.

The Diagnosis of Liver Disturbance by Functional Tests.—In those cases without a history or objective evidence of icterus, enlarged liver or spleen, ascites, unexplained pruritus or hematemesis:

1. *Syphilis.*—Candidates for antileutic therapy might well be submitted to liver function studies. Kerr and his associates have demonstrated that of ninety in whom the rose bengal excretory test was instituted prior to treatment for syphilis, 18 or 20 per cent showed hepatic dysfunction. In four, the disturbance became pronounced during arsphenamine administration. This incidence of subclinical hepatitis and involvement subsequent to therapy might have been higher were multiple tests measuring multiple functions employed. Thus, the hippuric acid test will sometimes show a disturbance in the detoxifying power of the liver when the excretory tests are normal. At any rate, with the knowledge of liver dysfunction prior to therapy such cases could be treated more cautiously, avoiding a more advanced hepatitis or cirrhosis in some of the relatively small number so affected.

2. *Systemic Infections.*—Liver function tests may be indicated in those having obscure symptoms months to years after having had measles, scarlatina, catarrhal or infectious jaundice, pneumonia or typhoid fever. These infections may result in a focal or diffuse necrosis with reparative processes resulting in varying degrees of permanent damage. In many, such evidence can be obtained only by multiple liver function tests: the Van den Bergh quantitative reaction, the bromsul-

falein excretory (using the 5-mg. per kilogram dose) and the hippuric acid (metabolic) tests. Only with knowledge of such disturbance obtained relatively early by liver function tests can protective measures be instituted.

3. *Hepatic Toxicoses.*—In those giving a history of the ingestion of inorganic arsenic, or cinchophen, possibly the imbibing of consistently large quantities of alcohol, and the administration of arsphenamine, mercury or chloroform, one or more of multiple liver function tests will often reveal abnormalities before any gross changes appear. Here, too, early diagnosis can be made only by liver function tests.

4. *Hepatic Dysfunction in Association with Other Diseases.*—Diabetes, rheumatoid arthritis, hyperthyroidism and sometimes ulcerative colitis are among the diseases in which changes of hepatic function are often encountered. Frequently, there is no bedside evidence of this, but such detection can be made by liver function tests. The latter are indicated when a complete study of such diseases is undertaken.

5. *Occult Hepatic Dysfunction or Occult Hepatitis.*—I have applied these terms to two groups of cases which I have encountered:

(*A*) Those giving a history of nausea with or without emesis, insomnia, and anorexia with epigastric distress of an apparently unexplained nature. This has also been referred to by James F. Weir.

(*B*) In some cases in which there is no history of any of the etiologic factors in hepatic disease, and no nausea, vomiting or anorexia, but which show inadequately explained symptoms referable to the digestive tract. These cases are often designated as "functional dyspepsia."

In these groups there are aberrations in the indirect quantitative Van den Bergh reaction (elevated, without clinical icterus), bromsulfalein test or hippuric acid test—sometimes in more than one—indicating hepatic involvement. The diagnosis can be established only by the introduction of these tests, which are indicated when the complaints referred to cannot be otherwise satisfactorily explained. Appropriate therapy often results in transient or permanent improvement as gauged by the tests; sometimes the disturbance remains stationary or progresses.

Uses in Differential Diagnosis of Jaundice.—(*A*) The differential diagnosis between *intrahepatic* and *extrahepatic* jaundice is important particularly because the former involves medical management and the latter usually requires surgical intervention. Intrahepatic jaundice is not always painless and extrahepatic jaundice may not be painful or may be only slightly so. In jaundice of sudden onset—that is, *acute jaundice*—the following tests are used simultaneously and may assist in the differentiation between these two types: the galactose tolerance, blood phosphatase and cholesterol partition. These should not be limited to single determinations; they should be repeated approximately three times during the course of a week during any period of jaundice. When fully equipped laboratories are not available to carry out the elaborate chemical procedures involved in the cholesterol and phosphate determinations, the simple galactose tolerance test if properly done early in jaundice probably remains the most reliable single laboratory method in the differentiation of intrahepatic from extrahepatic jaundice.

Galactose Tolerance Test.—Shay and Fieman state: "We have repeatedly stressed the fact that the physiology of the liver, with its tremendous reserve and excellent regenerative power, must put certain limitations upon any liver function tests and especially upon one concerned with so vital a function as carbohydrate metabolism.

"In obstructive jaundice not complicated by infection, the five hour output after 40 grams of galactose is below 3 grams, while in diffuse acute parenchymatous liver disease, the output is usually above 3 grams. But in obstructive jaundice with superimposed infection and associated parenchymatous damage, the output of galactose can exceed 3 grams. In the same way, a toxic hepatitis may be mild enough not to alter the carbohydrate function of the liver to cause an excretion of over 3 grams. During the ascent of a severe grade of toxic hepatitis, or in the recovery period, the galactose tolerance may again be normal. A repetition of the test and an interpretation in the light of the clinical and blood bilirubin findings will usually indicate the true state of affairs."

The Cholesterol Partition.—The liver is regarded as being the main excretory organ for cholesterol and regulates the

relative content of the blood in both cholesterol and cholesterol ester. In intrahepatic or toxic jaundice, the typical picture is a normal or moderately increased total blood cholesterol with a decrease in esters which results in a lowered ratio. Also, there seems to be a close inverse relationship in changes of blood bilirubin and cholesterol-ester ratio. Thus, in recovery, a rise in esters usually accompanies a fall in blood bilirubin.

The Phosphatase Level of the Blood.—This has been used as a means of differentiation. In obstructive jaundice, the units are usually above 20; readings below 16, provided one can rule out the recovery period of an obstructive jaundice, are in favor of a toxic or intrahepatic hepatitis.

(B) In *chronic jaundice,* liver function tests are usually not helpful in differential diagnosis. Their uses are indicated only for purposes of confirming the presence and extent of liver disease. Here the indirect quantitative Van den Bergh reaction indicating the degree of bilirubinemia, and the hippuric acid tests denoting involvement of the parenchyma are helpful.

In chronic jaundice, too, the test for *urinary urobilinogen* should be employed. Obstruction to the outflow of bile by a mass usually is complete; hence, repetitious urinary urobilinogen examinations will show its persistent absence. Obstruction due to stone or stricture of the common duct resulting in chronic jaundice usually is not complete, hence there is rarely complete or persistent absence of urinary urobilinogen upon repeated determinations.

Liver Function Tests as Aids to Prognosis and Therapy.—Once having determined liver dysfunction by one or more tests, repetitious examinations will indicate the stationary character of the disturbance or its regression or progression. In this way, too, the institution and influence of therapy as well as its cessation can be more accurately determined.

Hepatic function tests are indicated particularly in those who are to be submitted to surgery of the biliary tract. Here demonstrable deviations in hepatic function call for preoperative treatment directed toward the liver. Alterations in the tests may be the only manifestation of hepatic deficiency. Improvement of hepatic function as gauged by these tests has been instrumental in facilitating postoperative convalescence and reducing operative mortality.

Nothing has been said of the icteric index because the indirect quantitative Van den Bergh reaction measures the degree of bilirubinemia more accurately and is not influenced by carotinemia or hemolysis. Discussion of the albumin-globulin ratio and other changes in serum protein has been omitted here because from a practical standpoint these are rather late manifestations of liver disease and the tests referred to are simpler and usually give adequate information. The bilirubin excretion test which is much more sensitive than the bromsulfalein test has not been mentioned because of the fact that it is complicated and costly, although it is quite practical.

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